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CONTENTS

When and Why are Removable Tooth Replacements Indicated? When and Why are Multiple Abutments Indicated?	167	Allison G. James
Gingivitis	173	Balint Orban
Oral Health Conditions Among Army Personnel at the Army Engineer Center	180	Sigmund Stahl Alvin L. Morris
Periodontal Disease: Prevalence and Incidence	185	C. D. Marshall-Day, et al
Therapeutic Nutrition for the Periodontal Patient	203	Abraham E. Nizel
Sodium Silicofluoride as a Desensitizing Agent	208	Walter C. Stout
The More Important Advances in Periodontal Research and Their Clinical Implications	211	Donald A. Kerr
Discussion of Dr. Kerr's Paper	215	Robert Kesel
An Evaluation of the Peripheral Giant Cell Tumor	216	Richard L. Phillips William G. Shafer
Root Anomalies	222	Duncan Blades
Dynamic Equilibria of Oral Tissues III. Effects of Acute Starvation on Cytochrome Oxidase and Succinoxidase Activity of Oral Tissues	223	Philip Person George W. Burnett
Editorial	228	
The Distribution of Mitoses in the Epithelium of the Interdental Papillae of the Rat Molar	229	Christine M. Hirt, et al
"Irvangeline"	232	Paul A. Boyle
The Relationship of Blood Catalase Activity and Periodontal Disease	233	Harold R. Englander, et al
Announcements	179, 227, 235, 236-240	

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Thirty-ninth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Sheraton-Palace Hotel, October 1955

San Francisco, California

JUSTIN D. TOWNER, D.D.S., F.A.A.P., M.I.C.D.

Dr. Justin D. Towner is one of the most popular and respected members of the American Academy of Periodontology. He served as President of the Academy in 1928 and was the originator of, and now perennial chairman of the Round Table Luncheon Program of the Academy. He was graduated from Vanderbilt University in 1898 and while he is now in his fifty-eighth year of practice, he still maintains a daily ten hour office schedule.

He is an organizer, inventor, author and a teacher. His efforts as co-organizer and Dean of University of Memphis, College of Dental Surgery instigated the merger of two dental and four medical institutions known as the Memphis Medical Center, University of Tennessee. He instituted what was then, 1909, thought the first Department of Oral Hygiene, Prophylaxis and Periodontia and taught these related subjects twenty-four years. The Tennessee State Dental Association recently complimented Dr. Towner on the fiftieth anniversary of his presidency and fifty-seventh consecutive attendance.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FIFTY-FIVE

When and Why Are Removable Tooth Replacements Indicated? When and Why Are Multiple Abutments Indicated?

by ALLISON G. JAMES, D.D.S., *Beverly Hills, California*

THE purpose of any bridge, fixed or removable, should be to preserve the remaining teeth, to restore masticatory function, to restore phonetics, to improve esthetics, and to give comfort to the patient. While the latter four qualifications have varying values in each individual case, it appears that unfortunately the first purpose—that of preserving the remaining teeth—frequently receives the least consideration.

The following possibly over-simplified explanation has been found most effective with patients, as it presents graphically to them the principles involved.

The simplest bridge replacement to be considered, one or two teeth supported by an abutment tooth at each end of the span, may be likened to a hitching rail supported by a post at either end. If these posts are heavy and deeply set in hard, firm ground a great deal of sidewise pushing and pulling will not loosen the posts in the ground. If, however, the posts are not deeply set, and furthermore the ground is soft and soggy, the same lateral stresses soon will loosen the posts in the soil. Obviously, two posts at one end of this rail—or better still, two posts at each end of this rail—would improve the situation somewhat. But more effective still would be to place a rail at right angles to the first, running from the post at one end to a post or posts set in the soil some distance away. Expanding this three-cornered rail arrangement to a square corral, with perhaps additional supporting posts at the corners or at intervals in the side, completes the analogy. In these terms, consideration of the supporting bone structure from roentgenographic evidence becomes intelligible.

In accordance with the above, when it is necessary to go to the opposite side of a dental arch to obtain stability for the replacement of teeth missing on one side, then a removable type of appliance is indicated. (Fig. 1a,b) If the stability suggested by the corral, or right-angle rail described above, is to be achieved, it would appear that the most practicable attachments possible for a removable appliance are internal precision types. It has been found most expedient in the writer's practice to design internal attachments to fit the need of the individual case, and to use definite locking devices, such as Neurohr latches (Fig. 2), spring

Read at the fortieth meeting of the American Academy of Periodontology, in Miami, Florida on November 4, 1954.

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Fig. 1

Upper: Maxillary partial denture designed to retain 5 remaining sound natural teeth: the 2 central incisors, 2 molars on the right, 1 molar on the left. The horizontal spring wire shown snaps into a horizontal notch cut in the cingulum of the left maxillary incisor three-quarter crown. The original design anticipated that the line of insertion of the anterior attachments would preclude any tendency of the anterior portion of the bridge to drop but was inadequate; therefore this spring wire was added after testing in the mouth.
Lower: Restoration in place.

locks (Fig. 3) and so forth. This is particularly true of short bite cases, where precision internal attachments dependent upon friction for their resistance to displacement are highly unsatisfactory. The chief reason for the positive latch arrangement has been the excessive wear which soon occurs on the frictional type of attachment, resulting in the necessity for increasing the tension from time to time, and in the types most easily available today this results in breakage of the leaf sooner or later.

Where multiple abutments at a given point are used, it has been found advisable to use long dowels, with the male portion on the mesial aspect of the distal abutment of a pair, locking into the female slot on the distal of the next anterior abutment. (Fig. 4) A small set screw can be used if there is any reason to believe that the relation of the two abutments will not be maintained by this measure. (Fig. 5) It has been found, however, that when the dowels are sufficiently long with a counter-sunk portion at the gingival, that is, a well in the most gingival portion of the female slot, there is no noticeable change over a period of many years. This method allows conservative cavity preparation for the preservation of tooth structure and esthetics, which is not always possible where rigid solder joints between the two abutments are used. Furthermore, experience indicates that the slight amount of individual movement which this device

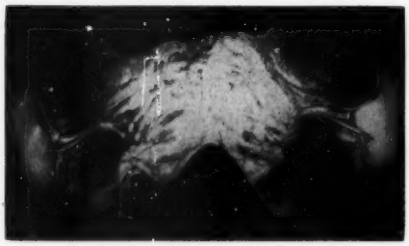

Fig. 2

Modified Neurohr latches. The vertical portion of each latch snaps into a notch prepared in the abutment casting.

allows between such abutment teeth is beneficial. It is always well to remember that the relation of the tooth to the alveolar process is that of a joint, and has the same elements in that articulation or joint that all other joints of the body do, including movement, even though it is exceedingly minute. If for any reason it is necessary to place the male dowel upon the distal surface of an anterior abutment, locking into the female slot in the mesial of the posterior abutment, then by all means the set screw at right angles should be employed, since this relationship tends to cause the dowel to be gradually displaced occlusally. (Fig. 6a, b)

Since it is possible for a patient to manipulate only two latches in a given

Fig. 3
A form of spring lock utilized to offset tendency to occlusal displacement. The protuberance or button at the end of the lingual half clasp snaps into a prepared receptacle or dimple.

appliance, it has been necessary to develop other types of attachments where several are to be used. A special posterior attachment which is self-locking and positive in action was developed some years ago and has proved eminently successful.[1] It is most applicable to short bite cases where there is not undue length of the anterior abutment requiring an elongated pontic just distal to it, since it must be removed on an arc with a radius centered on the posterior attachment. (Fig. 7)

A case which utilizes two of these self-locking posterior attachments and four anterior attachments is shown in Fig. 8a, b. This is an entirely tooth-supported partial replacement. This partial has been in use now something over seven years, and the abutment teeth remain firm.

In Fig. 9a, b is shown a case of a patient now 77. At the time this case was made the patient was 72. The remaining teeth in the arch, full complement in the maxillary arch and all except these missing anteriors in the mandibular arch are firm. At the time the appliance was constructed there was marked movement in all of the mandibular teeth. Some of this had been brought about through the wear of a tem-

Fig. 5
Two interlocking abutment castings are shown with the set screw described which tightens into a notch cut in the dowel on the distal casting. These castings are as removed from the investment and before polishing. After cementing the abutments to place and tightening the screw it will be cut off flush and polished even with the surface of the casting.

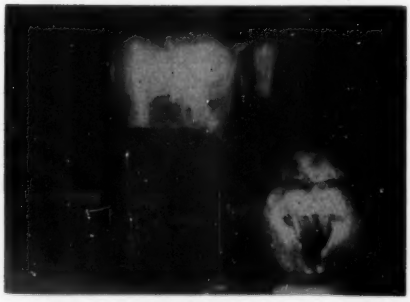

Fig. 4
Two interlocking abutment castings before final finishing down; illustrates the length of the dowel with the countersunk portion.

porary acrylic partial. Note that in this case there are four attachments used to give a four-post effect to the appliance.

It is always a moot question as to when a removable bridge becomes a partial denture. It is probable that any appliance utilizing soft tissue for some support should be considered as a partial denture. Figs. 10a, b, 11a, b show two partial dentures which are also removable bridge prostheses since they are both tooth-supported in the anterior portion, and use distal extension replacements in both maxillary and man-

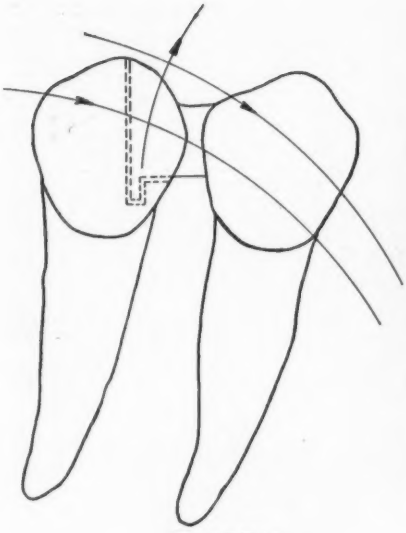

Fig. 6A

Illustrates the dislodging effect of mesial drift upon a dowel attachment between 2 abutment teeth when the receptacle is placed upon the distal attachment. Only the shearing effect of the distal surface or the male portion against the mesial wall of the receptacle offsets this displacing force. Under these conditions a locking screw is definitely indicated.

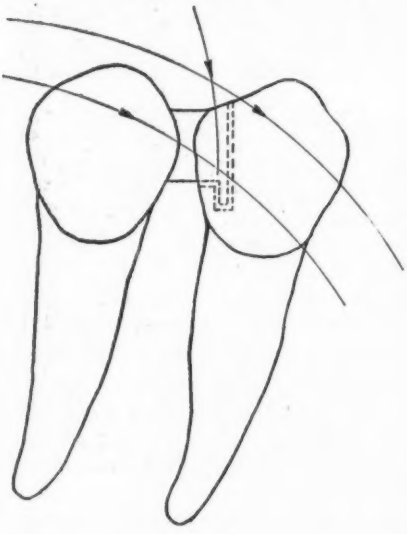

Fig. 6B

Illustrates the resultant force when the attachment is reversed under the same circumstances. The mesial drift tends to pull the dowel down into the receptacle progressively tighter and tighter.

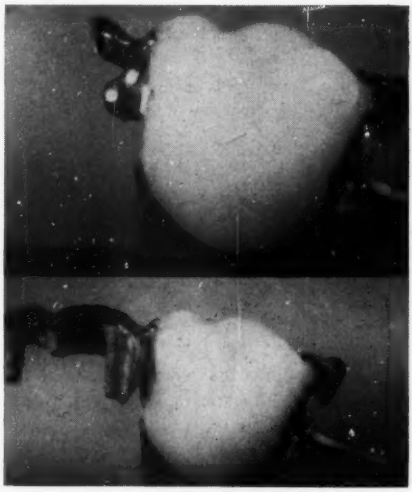

Fig. 7

Upper: Distal attachment designed to rotate into effective lock.
Lower: Abutment inlay and attachment in closed relation. Without occlusal rotation no displacement in any direction is possible.

dibular arches. Since the distal extension type of replacement has been fully discussed in other places,[2,3] this may be passed over briefly, only saying that the fundamental which must be considered is a type of distal extension attachment which will return the saddle to its rest position automatically through inherent qualities of the partial itself, following displacement by masticatory forces. When this principle is properly applied, absorption of the alveolar ridge is not found, nor does soreness develop at the distal portions of the saddle, with consequent settling out of occlusion. In this particular case there are only eleven natural teeth remaining in the mouth. Each tooth in the arch on a side is locked into the one anterior to it. When the removable prostheses are in place, the remaining teeth in an arch are locked together mechanically so that they must operate as a unit. The distal extension portions of the appliances operate individually, that is, the saddles on one side may be displaced by occlusal stresses with-

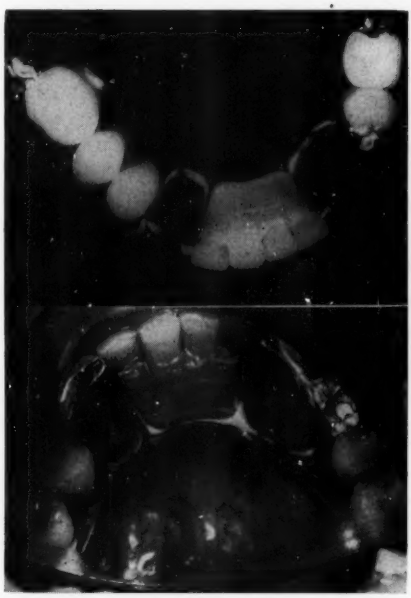

Fig. 8
Upper: Restoration utilizing 2 of the described posterior attachments with 4 dowel attachments in the anterior portion.
Lower: Intraoral mirror view of the appliance in position.

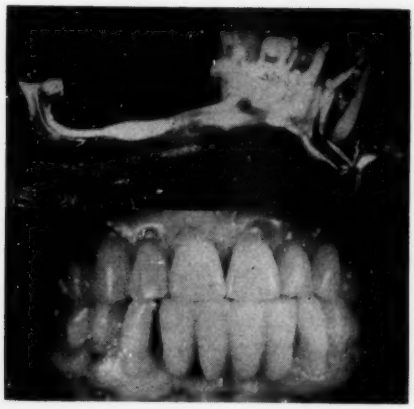

Fig. 9
Upper: Illustrates the 4-post principle in restoration of teeth requiring stabilization from both sides of the arch.
Lower: Appliance in place.

out affecting the saddles on the opposite side. With release of occlusal pressures the inherent spring quality of the appliance returns each saddle to its original rest position.

The most commonly encountered mandibular requirement is a distal extension on the right side and a single tooth replacement on the left side. Observations over twenty-seven years have led to the belief that people tend to do their most vigorous chewing on the side opposite the hand they use for eating. Therefore the periodontal health is most apt to be best on that heaviest working side. It has been possible in young people to pick out left-handers from the periodontal and soft tissue appearance. There are many local factors which may develop to change this habitual use of the opposite side for heaviest chewing, but it is noticeable that since most people are right-handed and eat with that hand, the largest

Fig. 10
Upper: Maxillary restoration illustrating utilization of spring locks, modified Neurohr latches, and self-recovering stress breakers with anterior portion rigidly tooth-supported.

Lower: Mandibular restoration illustrating utilization of spring locks, modified Neurohr latches, and self recovering stress breakers with anterior portion rigidly tooth-supported.

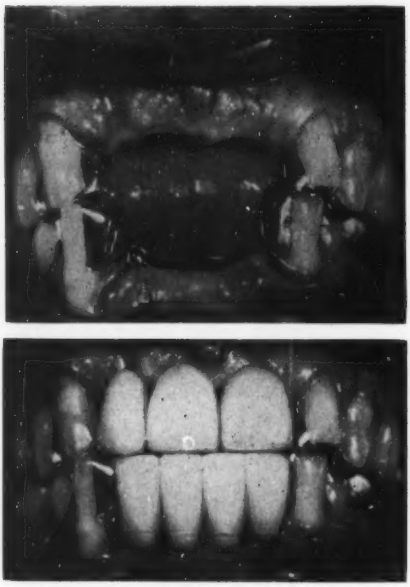

Fig. 11
Upper: Abutments of previously described prostheses showing receptacles for reception of replacements.
Lower: Restorations in position.

number of missing teeth is usually on the right side. Furthermore the periodontal destruction is noticeably greater on the less used side; which accounts for the greater loss of teeth on that side. By the time age sixty and beyond is reached this difference is perhaps not so noticeable, particularly as traumatic forces tend to come into greater play with excessive wear of the most used teeth.

In conclusion, then, it may be said:

1. Replacements of missing teeth which require stabilizing on the opposite side of the arch for the benefit of the abutment teeth should be removable in type.

2. The larger the number of abutment teeth which may be used, the more stabilizing the effect on the whole masticatory mechanism.

3. Interattachments of abutment teeth which do not preclude all individual movement are more beneficial than rigid soldered interattachments.

4. Where distal extensions are necessary, the type should return the saddle to rest position through inherent quality of the appliance; this reduces the tendency of ridge absorption almost completely.

5. If anterior portions of the dentition are to be replaced in conjunction with distal extensions a better result is obtained by making the anterior portion a part of the removable appliance than by utilizing fixed replacements in the anterior portion. This method produces an effective counterbalance and reduces stresses on the distal abutment teeth. Furthermore, it frequently allows better esthetics in anterior replacements where ridge resorption has been marked.—*409 North Camden Drive, Beverly Hills, California*

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Gingivitis*

by BALINT ORBAN, M.D., D.D.S.,** *Colorado Springs, Colorado*

PROPER management of such complicated conditions as periodontal diseases requires first that a reliable differential diagnosis be made, based upon a thorough clinical observation guided by a sound knowledge of pathology and an understanding of any possible systemic involvement.

Gingivitis is an inflammatory condition of the gingiva. The gingiva is that part of the oral mucous membrane which surrounds the teeth. We can differentiate between the free gingiva and the attached gingiva. The free gingiva is that part which forms the soft tissue part of the gingival sulcus or pocket. It extends from the margin of the gingiva to the bottom of the sulcus or pocket. On the outer surface, a fine groove, the free gingival groove, often separates the free from the attached gingiva. The attached gingiva is firmly anchored to the surface of the tooth and crest of the alveolus. This part of the gingiva shows a stippled appearance on its surface. The attached gingiva is separated from the rest of the oral mucous membrane by a festooned line, the muco-gingival junction. The clinical appearance of the gingiva is a reflection of the underlying structures and this, in turn, is governed by functional influences.

The attachment of the gingiva to the tooth is twofold—first by epithelium, the so-called epithelial attachment, and secondly by collagenous fiber bundles of the connective tissue which extend from the cementum into the free gingiva toward one side and to bone on the other. It is due to the presence of these heavy collagenous fiber bundles that the attached gingiva appears stippled. Since the oral mucous membrane is attached to a hard, calcified structure, there is the inevitable presence of a gingival sulcus. In ideal cases, this sulcus can be extremely shallow. On the average, it is 1 mm. to 2 mm. in depth. Gottlieb made the remark many years ago that "the shallower the gingival sulcus, the better it is," the reason being that the oral cavity is the natural breeding place for organisms. These organisms, together with organic matter that is present in the saliva, such as mucin, epithelial cells and leukocytes, deposit in the sulcus by the nature of its capillary suction. These organisms which are always present will produce toxins, leading to a tissue reaction in the connective tissue of the gingiva which is characteristic of inflammation. A mild inflammatory condition of the gingiva is always present and can be considered as a normal process, as it is not a disease, but a reaction to an irritant. If this irritation develops to such a degree that clinical symptoms appear, the diagnosis is "gingivitis."

In the diagnosis of gingivitis, one must proceed systematically. We *first* are interested in whether or not the gingival inflammation is localized or generalized. The *second* observation has to tell if the inflammatory process affects the papillary, marginal or attached part of the gingiva. *Third*, decide whether an acute or a chronic process is present. *Fourth*, we must observe the clinical features; if the disease has led to a hyperplastic type of gingivitis, or if it has resulted in ulcerations. If the latter, are the ulcerations limited or extensive; are they

*Bernhard Gottlieb had submitted a partially finished paper under the title of "Gingivitis" a few weeks prior to his sudden and untimely death. The illustrations were prepared for this paper, but the manuscript was never finished. The editor asked B. Orban to prepare the manuscript because most of the photomicrograms were made by him and published in a book by Gottlieb and Orban in 1933 in Berlin. Part of this book was later translated into English by M. Diamond and published by the Macmillan Co. in 1938 under the title, "Biology and Pathology of the Tooth and its Supporting Mechanism."

**The Colorado Dental Foundation.

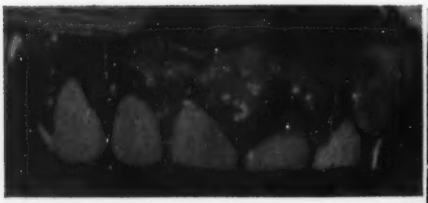

Fig. 1
Generalized, hyperplastic gingivitis, localized mainly to the papillary gingiva.

free of necrotic tissues or do they show a so-called pseudomembrane on the surface. Is there pus escaping from the pockets (purulent inflammation) or does it show any hemorrhage (hemorrhagic exudate). After it has been established what the clinical appearance of the gingiva is, we come to the *fifth* consideration, establishing the possible etiology of the disturbance. In general, any gingivitis may be due to local or systemic factors. One could safely make the statement that there is no case of gingivitis that would be caused by either local or systemic factors alone. There is always a combination of the two present and it is up to our judgment, after thorough investigation, to decide how much we have to consider one or the other. It would be far beyond the scope of this paper to try to illustrate and describe all types of gingivitis. Just a few examples will be shown and discussed.

A generalized, hyperplastic type of gingivitis is present in Figure 1. The enlargement of the gingiva is mainly localized to the papillae. The marginal gingiva is much less affected. The biopsy of the gingiva revealed a chronic process (Fig. 2). The inflammatory infiltrate consists almost exclusively of plasma cells. No extensive local irritation could be found in this case. No systemic conditions were found that could have been responsible. The diagnosis thus, is: Generalized, papillary, chronic, hyperplastic gingivitis of idiopathic nature. Frequently, such a hyperplastic gingivitis develops in adolescent children, combined often with mouth breathing.

Fig. 2
Biopsy specimen from generalized, hyperplastic gingivitis (Fig. 1). A: Epithelium, showing acanthosis, hydropic degeneration, lack of keratin layer. B: Lymphocytic and plasma cell infiltration in connective tissue.

The most characteristic and common acute type of gingivitis is the "Vincent's type" of ulcerative gingivitis. This can be either localized or generalized, most frequently the former. It is papillary in many instances, appearing as if the tips of the papillae might have been cut off. However, it occurs on the marginal part of the gingiva, too. Necrosis of the papillae is microscopically characteristic (Fig. 3) and the inflammatory reaction is acute, characterized by the presence of polymorphonuclear leukocytes.

Acute ulcerative gingivitis is not necessarily of the Vincent's type. It can occur in cases with moderately deep pockets; it can be an acute flare-up of a chronic process due to some local irritation such as an injury by the toothbrush, tooth pick or food impaction. Pus is seen in such cases seeping from the pocket area and micro-

Fig. 3
Autopsy specimen of a case of necrotizing, ulcerative gingivitis (acute) of the Vincent's infection type. A: Necrotic tissue. B, C: Cemento-enamel junction. D, E: Bottom of gingival pocket. F: Acute inflammation of connective tissue. H: Alveolar crest. K: Borderline between necrotic and vital tissues.

Fig. 4
Autopsy specimen. Ulcerated gingival pocket epithelium. A: Bottom of pockets. B: Pus in the pocket. C: Connective tissue densely infiltrated by leukocytes. D: Connective tissue fiber bundles.

scopically very extensive leukocytic infiltration is in evidence with leukocytes migrating into the pocket from an ulcerated papilla (Fig. 4).

Chronic irritation due to, for example, carious lesions (Fig. 5) or broken down root surfaces can lead to polyp-like proliferation of the gingival papilla (Fig. 6). Clinically, such a proliferated papilla might appear as an "epulis," tumor of the gingiva. Microscopically, we find either a severe inflammatory reaction without any special characteristic features, or we might find a "pyogenic granuloma," an inflammatory growth in which the proliferation of capillaries is predominant. Such "pyogenic granulomas" are a typical but not exclusive feature of the so-called pregnancy tumor. In pregnancy, gingivitis of a special type is often seen. This fact leads to the assumption that pregnancy is one of the systemic conditions responsible for gingivitis. However, we know today that gingivitis does not occur more frequently during pregnancy than otherwise. It is true that gingivitis in pregnant women often takes a different form and appearance, clinically as well as microscopically, but pregnancy is not responsible for the gingivitis. It is responsible only for the different manifestations. Therefore, we consider pregnancy as a modifying factor in gingivitis.

The same can be said of most or all systemic conditions including Vitamin C deficiency, even in its most dramatic form of scurvy. Vitamin C deficiency will lead to a modification of the connective tissue

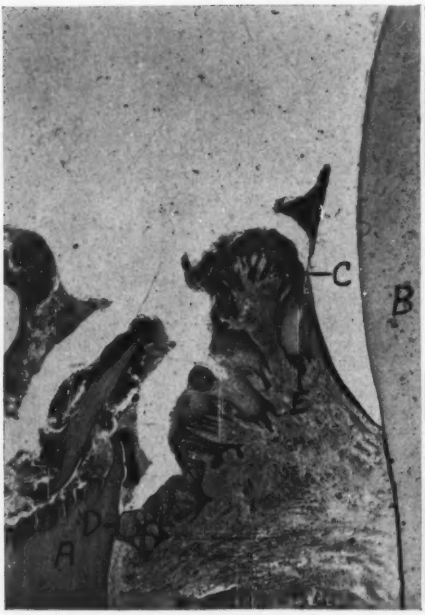

Fig. 5
Chronic gingivitis due to irritation by carious lesion. Autopsy specimen. A: Tooth destroyed by carious process. B: Healthy tooth. C: Bottom of pocket on intact tooth. D: Bottom of pocket on carious tooth. E: Proliferating epithelial ridges.

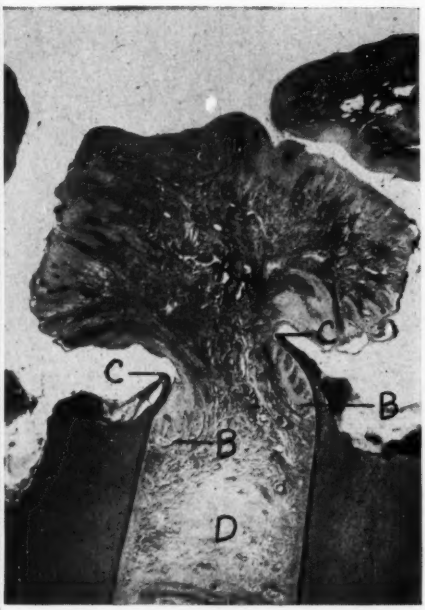

Fig. 6
Autopsy specimen. Polyplike proliferation of gingival papilla, due to irritation by root fragments. A: Gingival "polyp." B: Epithelial attachments. C: Root fragments. D: Transseptal fibers.

ground substance—a possible depolymerization of the long chains of glycoproteid molecules. This factor will be responsible for the capillary hemorrhage, extensive edema, acuteness of reaction. However, a local irritating factor is the one which will produce primarily the gingivitis. The tissue reaction that was produced by the deficiency might predispose the individual to infection or to a more severe local tissue response.

In certain cases, inflammation will become more or less stationary and chronic in character. In some of these instances, an ulcer might also develop at the base of the pocket; some leukocytes might be present in the tissues as well as in the exudate, but the bulk of the inflammatory infiltrate consists of lymphocytes and plasma cells (Fig. 7). In some such cases,

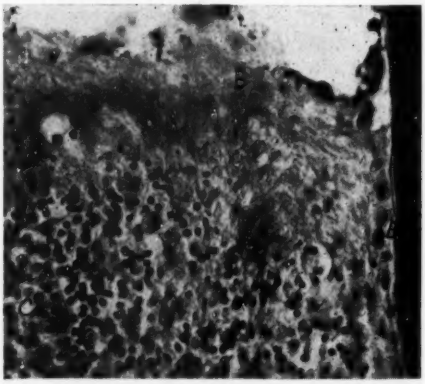

Fig. 7
Autopsy specimen. Chronic ulcerative gingivitis. A: Epithelial attachment. B: Necrotic surface of pocket epithelium. C: Lymphocytes and plasma cells.

Fig. 8. Autopsy specimen. Chronic irritation due to calculus deposits compressing interdental papilla. A: Cemento-enamel junction. B: Exposed cementum. C: Calculus. D: Epithelial attachments. E: Thin pocket-epithelium near the bottom of pocket. Epithelial ridge proliferation, chronic inflammation in connective tissue.

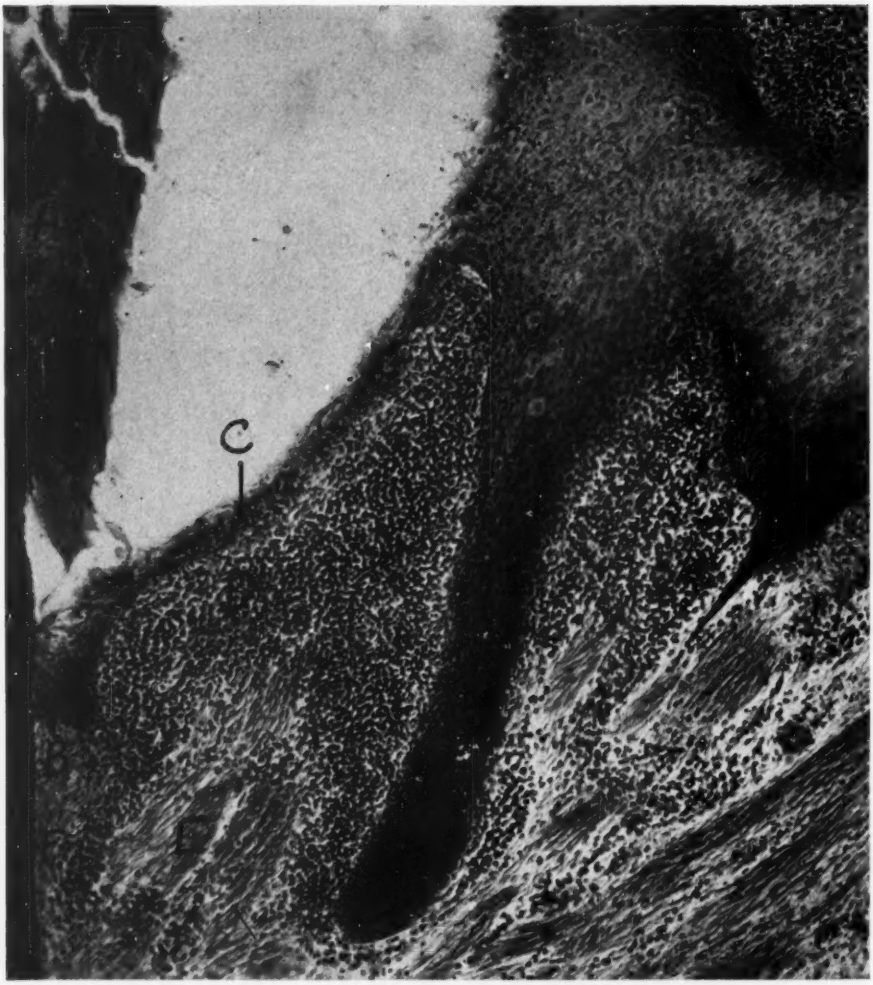

Fig. 9. Autopsy specimen. Chronic gingivitis. A: Calculus. B: Epithelial attachment. C: Thin pocket epithelium. D: Chronic inflammatory process (mainly plasma cells). E: Connective tissue fibers. F: Inflammatory cells progress along the blood vessels into deeper structures.

extremely large calculus masses can be found in the pockets (Fig. 8), compressing the gingival papillae, causing a venous stagnation and maintaining a chronic irritation with accompanying chronic inflammatory reaction. Epithelial proliferation often occurs in such cases and long epithelial projections extend into the inflamed connective tissue. The presence of extensive masses of plasma cells (Fig. 9) is an almost constant feature of chronic gingivitis. The plasma cells follow the course of blood and lymph vessels and these inflammatory cell accumulations often extend into the alveolar crest leading to resorption of the bony crest.

It has often been claimed that in periodontal disease this chronic inflammatory tissue is infected. This claim has been thoroughly disproven and beyond any doubt, this tissue is only a reaction to the irritation originating in the pocket. What should be done with this tissue in treatment has been the subject of many heated discussions. Some men have claimed that in not removing the granulation tissue, we jeopardize a good result of treatment. Today, we know that by removal of this tissue, we can have possibly one advantage only—faster post-operative healing. However, it is questionable that one is justified in sacrificing tissue that is capable of complete regeneration in the hope of a few days of faster healing. If the cause of the inflammatory infiltration is removed, one can expect the disappearance of the infiltrate. One does not treat the inflammation, but the cause of the inflammation which, in most instances, is a local irritation by calculus deposits, faulty dental restorations, open contacts, food impaction. These factors are frequently present without causing extensive inflammatory reaction. The individual's tissue reaction, dependent upon systemic factors, may determine the extent and severity of the process. Treatment of the inflammatory reaction without removing the cause would not only be futile, but dangerous. The inflammatory process is a defense mechanism of the body to prevent bacteria and their toxins from penetrating into the system. Breaking down this defense barrier would not be desirable. This fact makes it also clear that vigorous brushing, massage of the inflamed gingiva is not desirable as long as the local irritation is not eliminated and a possible systemic background corrected. Massage, brushing, use of astringent drugs or mouth washes have their value only after the inciting factor has been removed and the acute phase has subsided. The only value of such "massage" is the increase of arterial circulation by opening up additional capillaries, leading to more rapid elimination of the inflammatory products that have accumulated after doing their jobs in this defense mechanism.

We can thus conclude that one of the most important curative measures in any type of gingivitis is removal of all irritants from the teeth and maintenance of good oral hygiene. Recognition of systemic factors, such as endocrine disturbances, nutritional or dietary factors, deficiencies, etc., are, of course, of great importance. However, eliminating or correcting these factors without proper local treatment will not lead to any satisfactory results. It is our conviction that seventy-five per cent of all gingivitis cases will completely recover by local treatment and maintenance of good oral hygiene. The remaining twenty-five per cent will need some systemic assistance in their treatment.

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ARPA TO MEET IN VENICE

The 14th Congress of ARPA Internationale will be held in Venice September 5-11, 1955. For information, write Dr. A. J. Held, Geneva, Switzerland.

Oral Health Conditions Among Army Personnel at the Army Engineer Center

by S. Sigmund Stahl, Captain, D.C., U.S.A.R.,* and Alvin L. Morris, First Lieutenant, D.C., U.S.A.R.,** *Fort Belvoir, Virginia*

ANNUAL physical examinations of personnel stationed at Fort Belvoir, Virginia, offered an opportunity to study the oral health conditions of a group of soldiers. This group was composed of both officers and enlisted men who came from various geographic areas of the country. An investigation was planned to determine:

1. If a difference exists in the extent and severity of gingival disease between officers and enlisted men.

2. If a difference exists in the extent and severity of gingival disease between men from different geographic locations.

3. The relationship between gingival disease incidence and age. The investigation also provided an opportunity to observe:

1. Average tooth loss of different ages and rank.

2. The frequence of occurrence of chronic white lesions on the oral mucosa.

3. The mouths of a small group of Negro soldiers.

METHODS AND MATERIALS

In order to obtain a baseline suitable for comparison with other studies, 1,153 white males, ranging in age from 17 to 49 years and having six or more natural teeth, were examined, by means of flashlight and tongue depressors, for evidence of gingival papillary and marginal changes and presence of gingival recession. Findings were recorded using a modification of the P-M-A Index.[1] This modification omits the value of A (attached gingiva), and includes an R value (presence of gingival recession). The examination chart used provides for the recording of the soldier's age, race, rank, home state, and length of service, in addition to the PMR score, tooth loss and presence of white lesions.

The gingival papilla (P) or the margin (M) of a given tooth were recorded when they exhibited any deviation from normal in the opinion of the observer. Gingival recession (R) was recorded when the cemento-enamel junction was exposed. The results are given as percentages of teeth affected by both gingival inflammation and recession. For example: The P value for a soldier with 23 teeth and 5 inflamed papillae was calculated as 23/5 or 0.22. The P value (0.22) multiplied by 100 then became the P value in per cent. Similar calculations were carried out for the M and R values.*

The incidence of white chronic lesions of the oral mucosa, such as leukoplakia, lichen planus, etc., was also recorded.

FINDINGS

Gingivitis Experience of the Group: Inflammation of the gingival papilla tended to increase with aging from an average of 35 per cent of papilla involved in ages 17 to 19 years, to 45 per cent of papilla involved in ages 40 to 49 years. The marginal inflammatory changes, as well as the presence of gingival recession, also increased with aging (Table 1).

A comparison was made of the findings between Officers and Enlisted Men and the

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*A detailed description for this calculation is reported elsewhere.[2]

TABLE I

Average Tooth Loss and Gingivitis Experience in 1153 White Males Ranging in Age from 17-49 Yrs.

Age (Yrs.)	No. of Men	Length of Service (month)	Average No. of Teeth Missing ±S.E.	Average Per Cent of P Involved ±S.E.	Average Per Cent of M Involved ±S.E.	Average Per Cent of R Involved ±S.E.	No. of White Lesions	
							No.	Per Cent
17-19	26	22.5	6.0 ±.25	35 ±2.8	32 ±2.9	07 ±1.5	3	11.6
20-29	774	31.0	5.5 ±.15	35 ±1.7	30 ±1.6	10 ±0.4	129	6.0
30-39	272	135.3	6.1 ±.25	40 ±2.8	32 ±2.9	20 ±2.5	68	4.0
40-49	81	185.8	8.0 ±.50	43 ±1.9	37 ±2.0	29 ±1.5	23	3.5
Total	1153						223	5.2

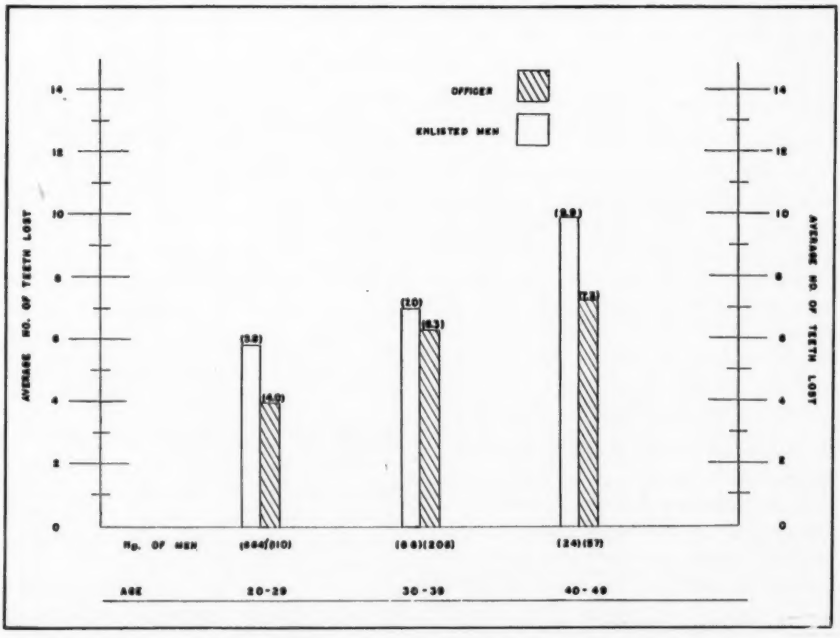

Fig. 1. Comparison of Average Tooth Loss Between Officers and Enlisted Men.

TABLE II

*Comparison of Average Tooth Loss and Gingivitis Experience between 373 Officers and 780 Enlisted Men Ranging in Age from 17-49 Years.**

	ENLISTED MEN						OFFICERS					
Age (Yrs.)	No. of Men	Average Length of Service (month)	Average No. of Teeth Missing ±S.E.	Average Percent of P Involved ±S.E.	Average Percent of M Involved ±S.E.	Average Percent of R Involved ±S.E.	No. of Men	Average Length of Service (month)	Average No. of Teeth Missing ±S.E.	Average Percent of P Involved ±S.E.	Average Percent of M Involved ±S.E.	Average Percent of R Involved ±S.E.
17-19	26	23	6.0 ± .42	35 ±1.83	32 ±1.79	7 ± .98		—	—	—	—	—
20-29	664	27	5.8 ± .08	35 ± .36	31 ± .35	10 ± .23	110	55	4.0 ± .18	34 ± .85	25 ± .78	8 ± .49
30-39	66	128	7.0 ± .28	45 ±1.22	42 ±1.22	29 ±1.11	206	143	6.3 ± .16	38 ± .67	29 ± .62	17 ± .52
40-49	24	176	9.9 ± .53	46 ±2.16	43 ±2.15	31 ±2.00	57	190	7.3 ± .31	42 ±1.32	34 ±1.26	28 ±1.20
Total	780						373					

*Computation of S.E. according to the formula S.E. $\%=\sqrt{\frac{pq}{N}}$

where p equals the fraction of P. M. and R. occurring
q equals 1−p
N equals No. of Men

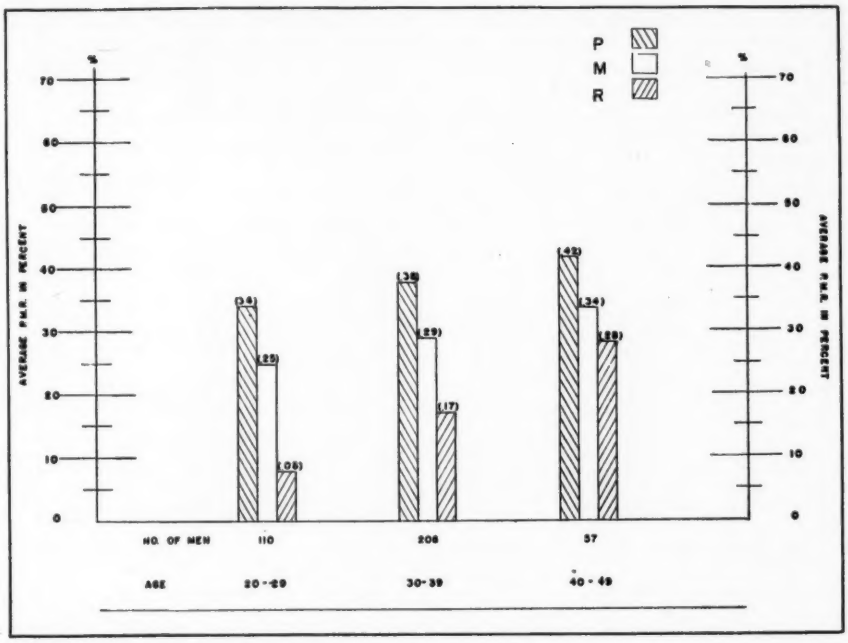

Fig. 2. Increase in Gingivitis Experience with Aging in Officers.

Standard Error of Difference was calculated for the two groups.** It was noted that:

1. Gingival papillary involvement was significantly lower in the officer group at ages 30 to 49 years.

2. Marginal gingival involvement was significantly lower in the officer group at ages 20 to 49 years.

3. Gingival recession occurred less frequently in the officer group at ages 20 to 39 years (Table 2, Figs. 2, 3).

Tooth Loss Experience of the Group: Tooth loss increased with aging from an average tooth loss of 6.0 teeth at the age of 17 to 19 years to 8.0 teeth at the age of 40 to 49 years (Table 1). Officers had a significantly lower tooth loss at ages 20 to 49 years (Table 2, Fig. 1).

Oral Conditions and Geographic Location: The United States was divided into four sections, Northeast, Central, Southeast and West, following the geographic division used by Massler and Ludwick.[3] The men were grouped according to their state of permanent residence. No significant difference was noted in gingivitis experience and tooth loss between the men coming from different geographic areas.

Findings in Negro Group Examined: A group of 150 Negro enlisted men, ranging in age from 20-39 years was also examined. No significant differences in gingival inflammation and tooth loss were noted between the Negro and the white groups.

Incidence of White Lesions: 223 of 5.2 per cent of the white examinees showed

**Statistical data was analyzed by Joe Zenewitz, B.S., Department of Dental Research, Army Medical Graduate School, Walter Reed Army Hospital, Washington, D. C.

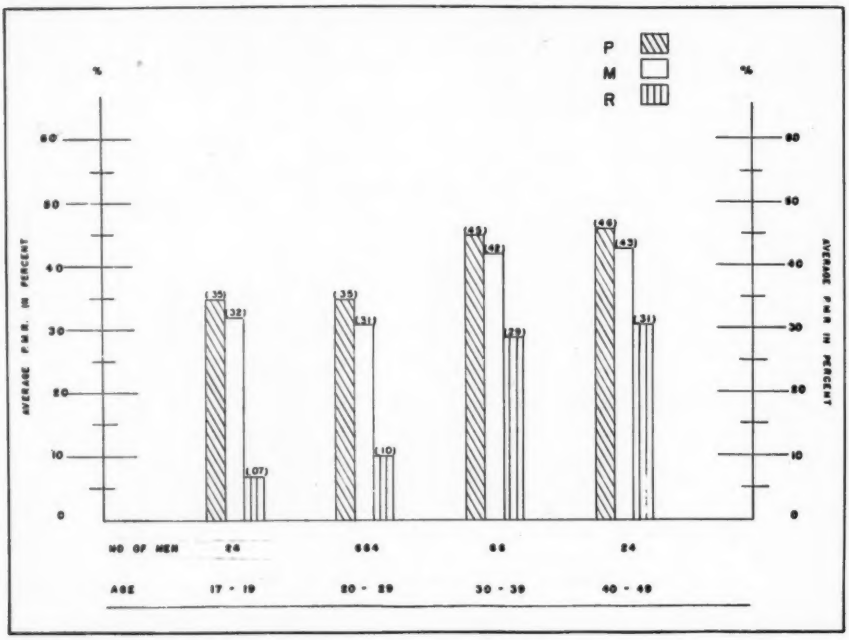

Fig. 3. Increase in Gingivitis Experience with Aging in Enlisted Men.

evidence of chronic white lesions on the oral mucosa (Table I).

Comment: The findings noted an increase in gingival inflammation and tooth loss with aging. These observations agree with those of Sandler and Stahl[4] who, using the same examining techniques, studied a group of hospitalized patients at the Veterans Administration Hospital, Brooklyn, New York. However, the inflammatory gingival involvement was greater in the young (20 to 29 years) soldier group than among the Veterans of similar age, which may be due to the poor oral hygiene practiced by soldiers in training. Analysis of the data also showed a significant difference in overall gingivitis experience and tooth loss between Officers and Enlisted Men which may reflect better oral health habits practiced by the officer group.

Neither tooth loss nor gingivitis experience was significantly different in persons from various localities, which agrees with the findings of Massler and Ludwick in their study of Navy Personnel.[5]

SUMMARY AND CONCLUSIONS

One thousand one hundred fifty-three white army personnel, ranging in age from 17-49 years, were examined to observe oral health conditions.

1. It was noted that gingival inflammation and gingival recession increased with aging. The officer group appeared to have a lesser degree of gingival involvement than the enlisted group.

2. Tooth loss increased with aging and was more commonly observed in the enlisted group.

3. The oral health conditions were similar in soldiers from different geographic locations.

4. The oral health conditions of 150 Negroes examined were not observed to be different from those of the white group.

5. 5.2 per cent of the group examined showed evidence of chronic white lesions on the oral mucosa.

The authors wish to thank Colonel G. J. Collins, D.C., and Lieutenant Colonel J. M. Murphy, D.C., for their assistance in arranging this study.

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Periodontal Disease: Prevalence and Incidence*

by C. D. MARSHALL-DAY, D.M.D., PH.D., RUSSELL G. STEPHENS, D.D.S. AND LAWRENCE F. QUIGLEY, JR., B.S., D.M.D., *Boston, Mass.*

IN recent years there has been a notable increased awareness on the part of the dental profession of the existence and importance of the periodontal problem. This is reflected in the greater emphasis placed upon the teaching of periodontology in our dental schools and in the increased number and diversity of studies reported in the current literature. For the most part, periodontal research has been concerned with the important aspects of etiology, pathology and treatment of the disease, but singularly little with its epidemiological characteristics. A review of the literature[1,2] indicates that much of our information on the prevalence and incidence of periodontal disease has been based on clinical impressions unsupported by adequate or reliable statistical data; yet a knowledge of the extent of the problem is a basic necessity in the prevention and control of the disease and in an appreciation of its public health aspects.

The present study was initiated in an attempt to throw some light on the epidemiologic characteristics of periodontal disease. As part of a more comprehensive study the present report will be confined to the prevalence and incidence of the disease in a representative sample of the male and female population in Boston.

*This investigation was supported by a grant from the Medical Research and Development Board, Office of the Surgeon General, Department of the Army, Contract No. DA49-007-MD-188. Study made in Tufts University School of Dental Medicine.

We wish to acknowledge the valuable counsel of Dr. Irving Glickman, and the assistance of the authorities in the following organizations: The Massachusetts Department of Public Health, the John Hancock Insurance Company, the Pratt Diagnostic Hospital and the schools and colleges named in this report.

Presented at the Thirty-Second General Meeting, International Association for Dental Research, French Lick Springs, Indiana, March, 1954.

REVIEW OF THE LITERATURE

Studies on the epidemiology of gingival disease, mostly on children and adolescents in the various age groups from 1 year to 18 years, have been much more numerous than on alveolar bone disease. The studies

by a number of workers on gingival disease prevalence have been discussed in an earlier report[1] and cover data from a variety of sources including the United States, Great Britain, Isle of Lewis, Sweden, India, New Zealand, Puerto Rico, Virgin Islands and Italy. The most noteworthy feature of the data presented is the wide range of prevalence reported even, in some cases, in the same geographical area. In many cases it is obvious that these differences are not actual but probably are related to a lack of uniformity in methods of assessment of gingival disease.

Studies on the prevalence and incidence of alveolar bone disease have been even less frequent and for the most part equally unreliable from the viewpoint of comparison of findings. A number of these studies have been carried out in other countries on population groups of very different ethnic origins and nutritional states, and some on groups that were potentially or frankly malnourished. These strictures apply to the reports of Schwartz in East Africa,[3] Marshall-Day and Shourie in India[4] and Dawson in Egypt.[5] Few of the epidemiologic reports relate to studies in the United States. The first of these by Black,[6] on 600 adults in the Chicago area revealed a prevalence of periodontal infections from 13 per cent at 20-24 years increasing to 88 per cent at 50 years and over. Miller and Seidler,[7] reported the need for periodontal treatment in 62 per cent of the males and 56 per cent of the females in 1003 hospital cases roentgenographically analyzed, and actual bone destruction increasing from 9 per cent in the 11-15 year age group to 97 per cent at age 41-45 years. From a study of 5,014 men in a Veterans Administration Dental Clinic in Chicago, Belting, Massler and Schour[8] reported that the incidence of all alveolar bone diseases combined reached a peak at 45-49 years of age, when 42 per cent of the male population was affected, and thereafter declined because of edentulousness. It might be considered that the last two studies were not entirely representative of the population as a whole since both were concerned with hospital patients, and one of them with men only.

In the absence of standardized diagnostic criteria and methods of assessment of periodontal disease it is virtually impossible in most cases to make a reliable comparison of prevalence and incidence figures in the studies conducted within or beyond the United States.

MATERIAL

Every effort was made to use material from a variety of sources in the Boston area in order to obtain, as nearly as possible, a representative cross-section of the population. Excluding those persons who failed to attend some phase of the examination or who had to be discarded for other reasons beyond our control, a total of 1,279 cases were examined. Of these, 92 were edentulous. The group comprised 935 persons participating in the Multiphasic Health Protection Programs conducted under the auspices of the John Hancock Insurance Company and the Department of Public Health; a total of 195 in the Sacred Heart High School; 86 freshmen dental students, and smaller groups of student nurses, Radcliffe College students and students from Boston College High School. Of the total, 633 were males and 646 females. The age range was from 13 to 65 years.

METHOD

In all cases a careful clinical examination was made by the same investigator (C.D. M-D), supplemented by models and Kodachrome films where indicated. The oral examination included a minute study of the gingivae with particular reference to color, size, shape, consistency and position. Pocket depth using a periodontal probe graduated in millimeters, tooth mobility, suppuration and supragingival and subgingival calculus were also recorded.

A 20-film intraoral roentgenographic survey including bite-wings was made in all cases for the purpose of studying the extent and distribution of bone loss, quali-

tative alterations in the appearance of the bony septa, trabecular patterns in areas unrelated to the teeth and the possible correlation of bone loss with gingival inflammation, pocket formation and tooth mobility.

Models of the teeth and gums were prepared to study cuspal relationships of individual teeth, overbite, overjet, plunger cusps and mutilations generally considered of etiological significance in the periodontal field, and for the purpose of analyzing occlusal disharmonies in relation to clinical and roentgenographic findings.

By questionnaire and interrogation data were collected also with regard to place of birth, family origin, previous dental history, habits such as bruxism, frequency of prophylaxes and toothbrushing, and tobacco consumption. Restorations, caries and tooth loss were also recorded.

Of the total group more than 700 cases between 19 and 65 years of age received a comprehensive medical examination at the New England Center and Pratt Diagnostic Hospitals. This examination was part of multiphasic health screening programs sponsored by the John Hancock Mutual Life Insurance Company and the Massachusetts State Health Department. This will be the subject of a separate report.

PREVALENCE OF GINGIVAL DISEASE

It is recognized that gingival disease may be characterized by an inflammatory condition, or a fibrous enlargement superimposed on a pre-existent inflammation. These conditions were recorded in terms of departure from the normal with regard to color, size, consistency and texture.

The clinical signs used for the assessment of the presence and severity of gingival disease were similar to those used by King,[9] by Marshall-Day and Shourie[10] and others: (a) Surface: disappearance of matt surface with reduction or absence of stippling effect. (b) Gum margins: swelling, color change from uniformly pale pink beyond the range of recognized variations from the normal, detachment from tooth surface with deepening of the gingival crevice; pocket formation and, in late stages sometimes suppuration and, more rarely, ulceration. (c) Papillae: swelling and color changes, which may be confined to the papillae or associated with similar changes in other parts of the gum margin; blunting of the normal pointed papillary tip; detachment from the adjacent teeth with interdental pocket formation. Hemorrhage may occur from any point of the gum margin, either spontaneously or on pressure. Only in the later and more severe stages of the disease will all of these signs appear together and then usually in association with alveolar bone disturbance. The signs will vary according to the type of disease studied. For example, it is possible to observe severe gingival enlargement with no detectable change in color or detachment from the tooth surface, and with little or no reduction in the normal stippling effect. In the group under examination the most common finding, especially in the younger age groups, was a change in density of the gingival tissue associated with extreme sponginess of the interdental papillae and a bluish-red coloration frequently confined to the papillae.

Table I presents the prevalence and degree of gingival disease recorded in the thirteen age groups between 13 and 65 years of age. These percentage figures indicate an extremely high prevalence at an early age, with a significant reduction in prevalence in the late teens and early twenties particularly marked in the case of the females. This phenomenon has been previously reported in this and other countries and has been variously ascribed to factors associated with the end of the period of puberty and to social factors concerned with a greater awareness of oral hygiene and aesthetic values at this age. In both male and female groups there was a sharp rise at age 23 years with a relatively constant high level of prevalence being maintained thereafter, and only a slight tendency to increase in the older age groups.

TABLE I

PREVALENCE AND DEGREE OF GINGIVAL DISEASE

Age Group	Number Examined			Gingival Disease Per cent			Degree: Slight Per cent			Degree: Moderate to Severe Per cent		
	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total
13-15	50	71	121	88	75	80	22	27	25	66	48	55
16-18	41	53	94	81	74	77	46	28	36	34	45	40
19-22	59	58	117	76	48	62	30	22	27	46	26	36
23-26	51	35	86	92	74	85	39	29	35	53	46	50
27-30	38	41	79	89	81	85	34	29	32	55	51	53
31-34	41	32	73	88	84	86	32	28	30	56	56	56
35-39	48	54	102	85	91	88	15	22	19	71	69	70
40-45	54	46	100	85	83	84	4	26	14	81	57	70
46-48	67	62	129	91	86	88	15	27	21	76	58	67
49-51	48	56	104	92	86	88	13	9	11	79	77	78
52-55	31	48	79	90	100	96	6	8	8	84	92	89
56-59	32	32	64	100	81	91	3	3	3	97	78	88
60 +	14	25	39	93	96	95	14	8	10	79	88	85
Total	574	613	1187	88	80	84	22	21	22	66	59	62

In ten of the thirteen age groups the males showed a greater prevalence than the females, with an overall average of 88 and 80 per cent for the males and females respectively. This relates also to the degree of severity of gingival disease. The figures for slight involvement were approximately the same for males and females, while 66 per cent of the males compared with 59 per cent of the females showed moderate to severe involvement. These findings are summarized in Fig. 1.

Table II presents the relative frequency of occurrence of the marginal and diffuse types of gingival disease and the relative frequency of gingival disturbance with and without color change. The figures emphasize the comparative absence of the diffuse type up to 40 years of age, and the consistently high incidence of the marginal type throughout all age groups. For males and females combined the average prevalence of the marginal type was 76 per cent and of the diffuse type only 7 per cent. The diffuse type was encountered with relative infrequency under 40 years of age. Gingival disturbance with color change occurred in 66 per cent and without color

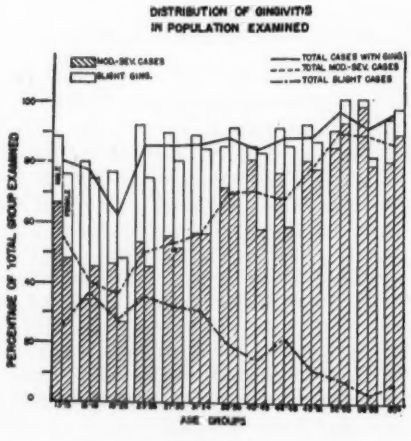

Figure 1.

TABLE II

INCIDENCE OF GINGIVAL DISEASE (Per Cent)

Age Groups	Classification			Marginal			Diffuse			With Color Change			Without Color Change		
	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total
13-15	50	71	121	88	72	78	—	3	2	74	69	71	14	6	9
16-18	41	53	94	80	72	76	—	2	1	66	62	64	15	11	13
19-22	59	58	117	76	48	62	—	—	—	64	33	49	12	15	14
23-26	51	35	86	90	74	84	2	—	1	82	63	74	10	11	10
27-30	38	41	79	89	80	85	—	—	—	76	59	67	13	22	18
31-34	41	32	73	83	78	81	5	6	5	68	69	68	20	16	18
35-39	48	54	102	81	89	85	4	2	3	71	78	74	15	13	14
40-45	54	46	100	68	76	72	17	7	12	69	61	65	17	22	19
46-48	67	62	129	76	76	76	15	10	13	60	65	62	31	21	26
49-51	48	56	104	65	75	70	27	11	18	69	63	65	23	23	23
52-55	31	48	79	77	85	82	13	15	14	58	75	68	32	25	28
56-59	32	32	64	69	75	72	31	6	19	87	62	75	13	19	16
60 +	14	25	39	93	72	80	—	24	15	64	76	72	29	20	23
Total	574	613	1187	79	74	76	9	6	7	70	63	66	18	17	17

change in only 17 per cent of the cases examined.

Table III outlines the regional distribution of gingival disease in the various age groups. At age 13-15 years the percentage of cases showing generalized disturbance was significantly greater than those in which the gingival changes were confined to localized areas. Reference to the line graph (Fig. 2) indicates that the lines merge at age 16, then show an equal distribution until age 22 years when the lines diverge showing a comparatively rapid increase in generalized gingival disease as age increases. Generalized gingival disturbance was consistently more prevalent amongst the males, whereas localized disturbance was in most age groups more prevalent in the females.

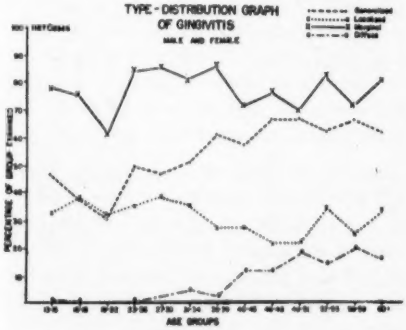

Figure 2.

The upper posterior regions of the mouth (74 per cent) were more commonly affected than the upper anterior regions (62 per cent). This was true for all age groups. In the lower jaw the posterior regions (64 per cent) were only slightly less affected than the anterior regions (66 per cent).

RECESSION AND POCKET FORMATION

Glickman[11] has clearly differentiated between the "actual position" of the gingiva, the level at which the epithelial attachment joins the tooth, and the "apparent position" or the level of the crest of the gingival margin in relation to the tooth.

TABLE III

DISTRIBUTION OF GINGIVAL DISEASE (Per Cent)

Age Groups	Number of Subjects M.	F.	Total	Generalized M.	F.	Total	Localized M.	F.	Total	Incidence in Various Regions: Upper Anterior M.	F.	Total	Upper Posterior M.	F.	Total	Lower Anterior M.	F.	Total	Lower Posterior M.	F.	Total
13-15	50	71	121	54	42	47	34	32	33	80	59	68	81	63	70	78	62	69	55	54	54
16-18	41	53	94	51	28	38	29	45	38	68	55	61	66	60	62	71	51	60	60	43	50
19-22	59	58	117	32	29	31	44	19	32	42	36	39	64	42	53	48	38	43	49	35	42
23-26	51	35	86	51	49	50	41	26	35	59	57	58	81	59	72	63	60	62	70	56	64
27-30	38	41	79	53	41	47	37	39	38	68	46	57	72	65	68	74	63	68	62	57	59
31-34	41	32	73	56	44	51	32	41	35	68	45	58	79	70	75	68	59	64	66	55	61
35-39	48	54	102	60	61	61	25	30	27	71	64	67	79	86	83	75	78	76	72	67	69
40-45	54	46	100	63	50	57	22	33	27	74	57	66	80	77	79	67	61	64	73	63	68
46-48	67	62	129	72	60	66	19	26	22	78	64	71	87	80	84	76	68	72	81	69	75
49-51	48	56	104	69	64	66	23	21	22	67	68	68	82	75	78	79	70	74	78	74	76
52-55	31	48	79	71	56	62	19	44	34	74	68	71	80	90	86	74	67	70	78	76	77
56-59	32	32	64	78	53	66	22	28	25	80	56	67	94	80	86	84	66	75	88	66	77
60 +	14	25	39	57	64	62	36	32	33	64	74	70	86	81	83	85	78	81	68	72	71
Total	574	613	1187	58	49	53	29	32	30	68	57	62	78	70	74	71	62	66	68	59	64

Recession or denudation of the root may therefore occur with or without visual exposure of the cementum or root surface. Exposure of the root is the result of apical movement of the gingival attachment and marginal crest of the gingiva. It is entirely possible that a pocket may occur through gingival enlargement without change in the actual position or level of attachment of the gingiva. Clinically differentiation between a true and false pocket is not always possible.

It is generally agreed that the average normal sulcus depth is approximately 2 mm but there may be significant differences within the normal range. For this reason, and in order to allow for some slight compression by the measuring probe at the base of the sulcus, penetration of the probe for a distance of 3 mm below the marginal crest of the gingiva was arbitrarily adopted as the criterion for pocket formation.

Table IV shows the results recorded in relation to recession and pocket formation. Section 1 of the table records the percentages of males, females and of the total group showing pocket formation due to coronal movement of the marginal crest of the gingiva through gingival enlargement, and/or apical movement of the epithelial attachment. In these cases there was no visual exposure of the root surface. In the youngest age group (13-15 years) a significantly higher percentage of males was affected (46:24 per cent). Thereafter there was some fluctuation as between the males and females until 27-30 years of age. Consistently from this age onwards a higher percentage of females was affected. The total figures decreased with age.

Section 2, Table IV records the percentage of subjects showing apical movement of the epithelial attachment and marginal crest of the gingiva with pocket formation and clinical exposure of the root. This was not encountered in the males until 19-22 years of age and in the females until 27-30 years of age. All age groups from 19-60

TABLE IV

INCIDENCE OF RECESSION AND POCKET FORMATION

Age Group	*1. Pocket formation with or without recession but no clinical exposure of root*			*2. Recession with pocket formation and clinical exposure of root*			*3. Recession with no pocket formation with clinical exposure of root*		
	Males	*Females*	*Total%*	*Males*	*Females*	*Total%*	*Males*	*Females*	*Total%*
13-15	46	24	43	—	—	—	—	—	—
16-18	27	36	32	—	—	—	—	—	—
19-22	66	47	56	2	—	1	—	—	—
23-26	69	66	67	8	—	5	2	—	1
27-30	55	63	59	16	7	11	8	—	4
31-34	59	62	60	24	12	19	7	6	7
35-39	46	61	54	31	30	30	12	4	8
40-45	22	46	33	54	37	46	7	7	7
46-48	16	39	27	79	42	61	3	10	6
49-51	17	27	22	75	59	66	6	11	9
52-55	16	31	25	74	65	68	10	2	5
56-59	19	19	19	81	66	73	—	13	6
60 +	7	8	8	79	80	79	14	12	13
	38	42	39	39	28	32	5	5	5

years of age showed a significantly lower figure for the females, with a steady increase for both males and females with increasing age.

Recession and root exposure by apical movement of the gingival crest but without pocket formation (Section 3, Table IV) was not encountered in the males until 23-26 years of age and in the females until 31-34 years of age. Thereafter no consistent pattern could be established, and of the whole group only 5 per cent of the males and 5 per cent of the females could be placed in this category. These findings are illustrated in the line graph (Fig. 3).

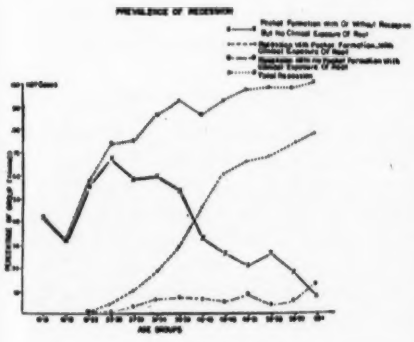

Figure 3.

ORAL HYGIENE

Included in the data derived from a questionnaire and by interrogation was information on frequency of dental visits, dental prophylaxes and brushings (Table V). It is recognized that information of this sort must be accepted with considerable reserve since it relies, to some extent at least, upon the memory and veracity of the individual. However it was considered of sufficient interest to report the findings in view of the possible bearing of these factors upon the incidence of periodontal conditions.

The average number of dental visits during the previous three years was 4.7 for

TABLE V

Age Group	Av. number dental visits last 3 years		Dental prophylaxes per year — per cent 2		1		<1		Brushing per day — per cent >1		1		<1	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F
13-15	6	6	28	37	26	44	46	19	40	66	54	32	6	2
16-18	6	10	17	36	44	45	39	19	41	87	54	13	5	—
19-22	6	6	29	21	34	45	37	34	85	88	14	12	1	—
23-26	6	6	27	11	33	40	40	49	67	69	31	31	2	—
27-30	6	6	16	20	61	37	23	44	68	73	26	27	6	—
31-34	6	6	15	25	29	38	56	37	44	59	56	41	—	—
35-39	4	6	19	28	54	46	27	26	42	63	56	37	2	—
40-45	4	4	37	41	35	37	28	22	37	67	61	33	2	—
46-48	4	4	48	55	25	26	27	19	45	66	52	34	3	—
49-51	4	4	38	36	27	34	35	30	58	70	40	30	2	—
52-55	3	4	61	58	13	27	26	15	57	77	35	23	7	—
56-59	2	3	37	50	13	22	50	28	60	81	34	16	6	3
60 +	1	3	29	44	29	24	42	32	29	84	58	12	13	4
13-60 +	4.7	5.4	31	36	33	37	36	27	53	73	44	27	3	—

the males and 5.4 for the females, with a reduction for both males and females in the older age groups. In 9 of the 13 age groups examined the females recorded a larger percentage having two dental prophylaxes per annum. Of all subjects examined, 31 per cent of the males and 36 per cent of the females reported two prophylaxes a year. In 10 of the 13 age groups the females recorded a larger percentage having one prophylaxis a year, the percentage for all age groups being 33 and 37 per cent for the males and females respectively. For less than one prophylaxis per year the position was reversed. In 11 of the age groups the males recorded a larger percentage, the figures for the entire group being: males 36 per cent, females 27 per cent.

Table V also records the percentage of subjects reporting more than one, one, and less than one brushing a day. Here again the females registered a better performance than the males. In all age groups the females reported a larger percentage brushing more than once a day, the percentage for all groups being females 73 per cent, males 53 per cent. In all age groups the males appeared to favor one or less than one brushing a day. The age of the individual seemed to have little bearing upon the frequency of dental prophylaxes. The best brushing performance was registered in the case of the females between the ages of 16 and 30 years and of the males between 19 and 30 years.

It is interesting to speculate as to the relative importance of better oral hygiene and more regular brushing habits of the females as factors in the demonstrated lower incidence of gingival disease.

INCIDENCE OF CALCULUS

It is recognized that the presence or absence of calculus at the time of examination is not a safe criterion for the rate or degree of calculus deposition in an individual case, since brushing habits and the time, frequency and adequacy of recent prophylaxes will have a direct bearing upon this finding.

Table VI shows the percentage of sub-

TABLE VI

INCIDENCE OF CALCULUS — TYPE AND DEGREE

Age Group	Per cent with demonstrable Calculus			Calculus Type (per cent)									Calculus Degree—Males and Females (per cent)					
				Supragingival only			Subgingival only			Both Sub and Supra			Supragingival			Subgingival		
	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total	Slight	Mod.	Sev.	Slight	Mod.	Sev.
13-15	30	34	32	20	14	17	—	8	5	10	11	11	21	6	—	10	6	—
16-18	46	32	38	24	23	23	5	8	6	17	2	9	23	7	1	11	4	—
19-22	69	59	64	37	47	42	3	2	2	29	10	20	54	7	1	17	5	—
23-26	65	57	62	25	31	28	6	6	6	33	20	28	38	17	—	17	16	—
27-30	58	66	62	29	22	25	5	12	9	24	32	28	42	10	1	26	9	1
31-34	80	63	73	29	25	27	7	3	6	44	34	40	51	14	2	27	15	3
35-39	81	89	85	29	31	30	8	9	9	44	48	45	45	30	1	25	28	1
40-45	85	74	80	19	30	24	9	4	7	57	39	49	46	22	5	29	22	5
46-48	81	61	72	24	19	22	9	15	12	48	27	38	37	18	5	26	18	5
49-51	73	68	70	19	21	20	13	9	11	42	38	39	36	20	4	25	21	4
52-55	90	65	75	23	27	25	10	2	5	58	35	45	44	22	4	24	22	4
56-59	91	91	91	7	38	22	13	13	13	72	41	56	47	20	11	42	16	11
60 +	79	88	85	36	32	33	—	12	8	43	44	44	44	28	5	20	26	5
Total	71	62	66	25	27	26	7	8	7	39	28	33	40	16	3	22	15	3

jects with clinically demonstrable calculus. The figures show a marked increase at 19-22 years of age and a further sharp increase at 31-34 years for both males and females, reaching a peak of 91 per cent at 56-59 years. The average percentage for males and females was 71 and 62 respectively. Twenty-six per cent of the total exhibited supragingival calculus only, with no significant difference between males and females. Cases with subgingival calculus alone were comparatively rarely encountered, the figures being 7 and 8 per cent for males and females respectively. After 30 years of age by far the greater proportion of subjects exhibited both supragingival and subgingival calculus. The percentage figures increased with age, the increase being most marked after 30 years of age. For all groups from 13 to 65 years the total average figures were: males 39 per cent, females 28 per cent. These findings are illustrated in the calculus type-distribution graph (Fig. 4), and the relative degree of calculus accumulations of both types for the various age groups in Fig. 5.

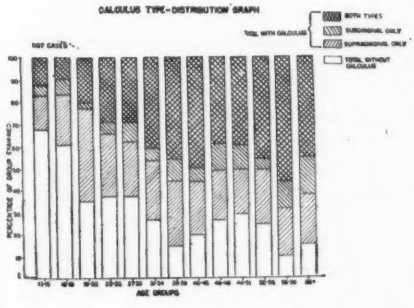

Figure 4.

PREVALENCE OF CHRONIC DESTRUCTIVE PERIODONTAL DISEASE

In the absence of an established "normal" bone-height baseline at various age levels, for the purpose of this study the condition of the alveolar bone was con-

TABLE VII

PREVALENCE OF CHRONIC DESTRUCTIVE PERIODONTAL DISEASE

Age Group	Number subjects examined*			Per cent with C.D.P.D.			Per cent C.D.P.D. with ging. dis.			Per cent C.D.P.D. without ging. dis.			Per cent with ging. dis. only			Per cent without ging. dis. or C.D.P.D		
	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.	Total	M.	F.
13-15	121	50	71	4	6	3	3	6	1	1	—	1	77	82	73	19	12	24
16-18	94	41	53	5	10	2	4	7	2	1	2	—	72	73	72	22	17	26
19-22	117	59	58	24	24	24	15	19	12	9	5	12	47	58	36	29	19	40
23-26	86	51	35	69	71	66	62	65	57	7	6	9	23	27	17	8	2	17
27-30	79	38	41	84	82	86	73	74	73	10	8	12	11	16	7	5	3	7
31-34	73	41	32	90	90	91	81	83	78	10	7	12	6	5	6	4	5	3
35-39	102	48	54	98	97	98	86	83	89	12	15	9	2	2	2	—	—	—
40-45	100	54	46	98	96	100	83	83	83	15	13	17	1	2	—	1	2	—
46-48	129	67	62	100	100	100	88	91	86	12	9	15	—	—	—	—	—	—
49-51	104	48	56	100	100	100	88	92	86	12	8	14	—	—	—	—	—	—
52-55	79	31	48	99	100	98	95	90	98	4	10	—	1	—	2	—	—	—
56-59	64	32	32	100	100	100	91	100	81	9	—	19	—	—	—	—	—	—
60 +	39	14	25	100	100	100	97	100	96	3	—	4	—	—	—	—	—	—
	1187	574	613	71	73	69	63	66	60	8	7	9	21	23	20	8	5	10

*Excluding edentulous cases.

sidered as "normal" if the alveolar crests were not resorbed to more than 2 mm below the cemento-enamel junction. An individual was considered as having chronic destructive periodontal disease if there was definite roentgenographic evidence of destruction of the alveolar bone surrounding one or more teeth. In the great majority of cases when periodontal disease was present a number of teeth were involved. Chronic destructive periodontal disease refers to periodontal disease with bone involvement, with or without clinical evidence of gingival disease. Only rarely was the disease encountered without accompanying gingival disturbance recognizable clinically. During the examination no attempt was made to classify the cases in accordance with the commonly accepted categories of periodontitis simplex and periodontosis.

Table VII records the prevalence of chronic destructive periodontal disease in its different phases in the various age groups. This disease was rarely found between 13 and 18 years of age, but the incidence increased rapidly from 24 to 69 per cent between the ages of 19 and 26 years. Thereafter the incidence steadily increased until 45 years of age when almost all subjects showed evidence of bone loss. The males (73 per cent) showed only a slightly higher figure than the females (69 per cent).

Bone loss without clinical evidence of ginvival disturbance was not a common finding (males 7 per cent: females 9 per cent), although the females from 19-35 years showed a greater prevalence than the males. Gingival disease without bone loss was confined almost entirely to the younger age groups, rapidly decreasing from 77 per cent at 13-15 years to 23 per cent at 23-26 years. The line graph (Fig. 6) illustrates some of these findings. It should be emphasized that the figures presented relate to both generalized and local-

ized bone loss. The condition was rated as generalized if the bone destruction was distributed throughout all or most of the mouth quadrants with some degree of uniformity, and localized if confined to one or two quadrants or to isolated teeth or groups of teeth. The high figure of 69 per cent with bone loss at 26 years represented 49 per cent localized and 20 per cent generalized. Figs. 7 and 8 show the degree and distribution of chronic destructive periodontal disease, with and without associated gingival disease. There was a consistent increase with age in the prevalence of generalized bone loss. At age 35-39 years 67 per cent were generalized and only 11 per cent localized. At age 60 all subjects were in the generalized category.

In the younger age groups the majority of the cases were classified as slight in degree, i.e., showing an early detectable departure from the normal. At age 23-26 years, of the 69 per cent with chronic destructive periodontal disease, 48 per cent were classified as slight in degree. Thereafter the percentage of subjects with moderate to severe degree of bone loss rapidly increased, and at age 35-39 years 43 per cent showed a moderate degree and 30 per cent a severe degree of involvement. This trend continued in the older age groups in which all subjects were recorded as having severe generalized bone destruction. The degree of severity was determined by the extent to which the retention of individual

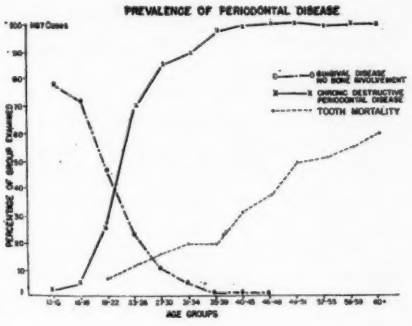

Figure 6.

teeth was jeopardized by bone loss.

Data were recorded with regard to the relative occurrence of the various types of septal shearing. By far the greater proportion of cases showed septal shearing of the horizontal type. A considerable number of individuals showed both horizontal and vertical shearing in different areas and relatively few showed vertical shearing alone. Following are the percentage figures for males and females in the three categories:

	Horizontal Shearing	*Vertical Shearing*	*Vertical and Horizontal Shearing*
	%	%	%
Males	65.3	1.6	18.6
Females	62.2	2.4	20.7

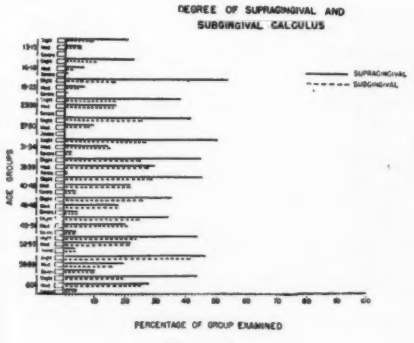

Figure 5.

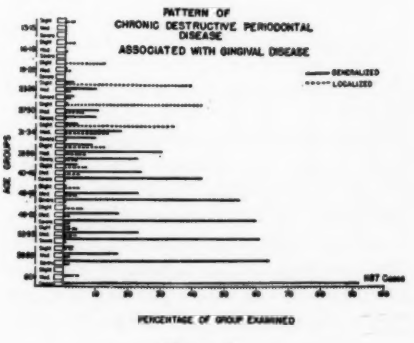

Figure 7.

TABLE VIII

DEGREE OF ALVEOLAR BONE RESORPTION

Age Group	Average "Resorption Figure"			Average Minimum Resorption			Average Maximum Resorption		
	Males	*Females*	*Total*	*Males*	*Females*	*Total*	*Males*	*Females*	*Total*
13-15	0.10	0.10	0.10	—	—	—	1.15	1.10	1.12
16-18	0.16	0.12	0.14	—	—	—	1.20	1.07	1.13
19-22	0.24	0.22	0.23	—	0.02	0.01	1.45	1.35	1.40
23-26	0.85	0.69	0.78	0.31	0.26	0.29	2.00	2.02	2.01
27-30	0.97	0.97	0.97	0.45	0.36	0.40	2.16	2.68	2.43
31-34	1.27	1.26	1.27	0.50	0.59	0.54	2.70	2.59	2.65
35-39	1.77	1.72	1.74	0.91	1.05	0.98	3.54	3.31	3.42
40-45	2.32	1.99	2.16	1.32	1.19	1.26	4.26	3.56	3.94
46-48	2.53	2.44	2.49	1.67	1.56	1.61	4.41	4.27	4.34
49-51	2.77	2.87	2.83	1.81	2.09	1.96	4.58	4.36	4.46
52-55	2.75	2.77	2.76	1.77	2.00	1.91	4.77	4.31	4.49
56-59	3.71	2.40	3.06	2.59	1.58	2.09	5.81	4.05	4.93
60 +	3.54	3.20	3.32	2.50	2.16	2.28	5.71	5.12	5.33

There was a slight but hardly significant tendency for the females to show a greater prevalence of vertical shearing alone and in combination with the horizontal type.

DEGREE OF ALVEOLAR BONE RESORPTION

Assessment of the degree of alveolar resorption was based on the degree of involvement of the interdental bony crests of the alveolus, as determined from a 20-film intraoral roentgenographic survey including bitewings. With certain obvious qualifications this method may be considered sufficiently reliable to give a reasonable accurate average resorption figure for each individual or age group.[4]

The degree of bone resorption was graded from 0 to 10. The rating "10" denotes complete loss of supporting bone as far as the root apex, "5" indicates loss of half the alveolar support and "1" denotes early detectable resorption. Intermediate numbers denote corresponding degrees of bone loss. The summation of the figures for each area examined gives the resorption score for each subject, and this total divided by the number of areas examined, the average degree of bone resorption or "resorption figure". Similarly the average resorption figure for any age group may be determined. By similar means the average minimum and maximum resorption for a group may be obtained from the lowest and highest degree of resorption noted in each individual.

These findings are recorded in Table VIII. Reference to the final column shows that the highest "resorption figure" found in any area for any individual in age group

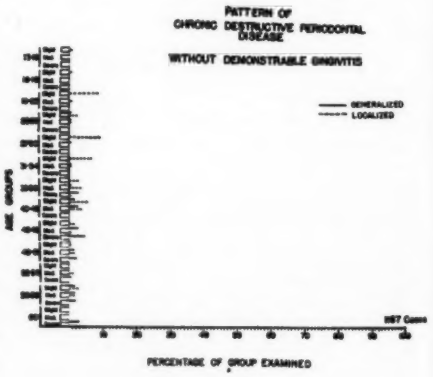

Figure 8.

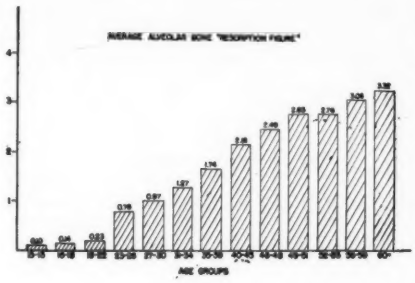

Figure 9.

13-15 was 2. This means that areas were found in which two-tenths of the supporting alveolar bone was lost. The highest resorption figure met with in any alveolar area in an individual between the age of 16 and 22 was 3. There was then a sharp rise to 6 in the 23-26 age group, the figure increasing gradually until age group 35-39. In this and all six older age groups areas were found where the supporting alveolar bone was resorbed as far as the root apex (resorption figure 10).

The first 3 columns in Table VIII show the Average Resorption Figure for the males and females and for males and females combined. Of particular interest is the more than threefold increase between the ages of 22 and 26 years, followed by a more gradual but steady increase in the older age groups. By age 46-48 an average of one-fourth, and by 60 years one-third of alveolar bone support was lost. The next two series of columns show the Average Minimum Resorption and the Average Maximum Resorption for each age group. The bar graph (Fig. 9) illustrates the progressive increase in the Average Resorption Figure in the 13 age groups.

In this connection it may be of some interest to compare these findings with those from a group of subjects examined under similar conditions by the same examiner (C.D.M-D.) in Northern India.[4] The alveolar bone resorption figures for the U.S.A. and India are compared in Fig. 10. It will be seen that chronic destructive periodontal disease has its incipiency at an even earlier age in East Indian subjects and progresses more rapidly and with a greater degree of bone loss in all age groups. The irregularities in the older age groups of the Indian line graph are doubtless due in part to the smallness of the groups and in part to the greater complication of edentulousness. Both curves would show a greater upward trend in the older age groups if it were possible to include a resorption figure for edentulous cases and for

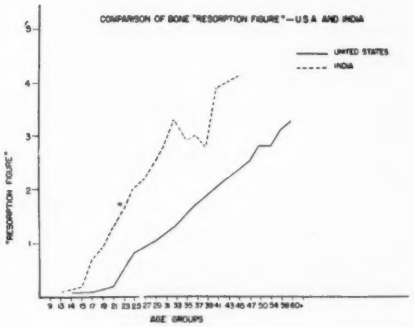

Figure 10.

all areas in partially edentulous individuals. This is indicated by the high tooth mortality figures in Table IX.

Also, periodontal disturbance in India was characterized by a much higher incidence of vertical shearing either alone or in combination with the horizontal type. The cause of the earlier and more severe periodontal breakdown in the Indian subject is not known. The group examined comprised mostly police constables selected for service largely because of good physical condition. It is therefore unlikely that they were undernourished. It is possible, however, that malnutrition was present in some degree, associated with a high carbohydrate diet but an inadequacy of proteins and other food factors. Also oral hygiene, if practised at all, was more likely to lead to traumatism of the gingivae due to the use of teased

TABLE IX

TOOTH MORTALITY

Age Groups	Number Subjects Examined	Teeth Lost Per Cent						Total Upper & Lower	Average Number Teeth Lost Per Subject	Per Cent Edentulous
		Upper			Lower					
		Ant.	Bi.	Mol.	Ant.	Bi.	Mol.			
19-22	117	1	4	11	0.5	2	15	6	2	—
23-26	86	3	8	22	1	5	27	12	4	—
27-30	79	3	13	31	4	12	32	16	5	—
31-34	73	11	17	32	2	13	37	19	6	—
35-39	102	6	16	37	1	9	38	19	6	—
40-45	105	19	33	53	6	20	56	32	10	5
46-48	143	24	43	60	11	22	61	37	12	10
49-51	118	44	54	69	18	35	70	49	15	12
52-55	107	39	55	69	27	43	72	51	16	26
56-59	81	48	63	72	26	43	77	55	17	21
60 +	53	48	63	78	34	58	82	60	19	26
Total	1064*	22	33	48	11	23	51	32	8	7

*Age groups below 19-22 years not included in table.

"dattan" sticks as a brushing agent.

TOOTH MORTALITY

To obtain a true picture of the conditions under examination the tooth mortality rate must be considered in conjunction with the morbidity figures. A record of the completely edentulous cases alone does not furnish sufficient information on tooth mortality, though this criterion was used in a recent investigation.[8]

Table IX shows the percentage of teeth lost for all reasons in the eleven age groups from 19 to 65 years. Figures are presented for tooth loss in relation to the upper and lower anteriors, bicuspids and molars, the percentage of teeth lost in the upper and lower jaws combined, the average number of teeth lost per subject and the percentage of completely edentulous cases. Tooth mortality in the younger age groups can reasonably be considered as due to causes other than periodontal disease; but the sharp rise in mortality at age 40-45 years to 32 per cent would seem to be related to the morbidity figures and the pattern of chronic destructive periodontal disease which becomes more generalized and more severe in degree by this age. Tooth mortality after 40 years increased rapidly. At 49-51 years 50 per cent of the teeth were missing, and 60 per cent by 60 years. The lower anterior teeth were most resistant, while the upper and lower molars showed the highest mortality in all age groups. By age 40-45, 53 per cent of the upper molars and 56 per cent of the lower molars had been lost. At this age 5 per cent of the subjects examined were completely edentulous and 26 per cent at age 60. The incidence of tooth mortality is illustrated in Fig. 6. The rapid rise in the mortality curve from 35 years onwards is significant.

POCKET FORMATION, TOOTH MOBILITY AND SUPPURATION

It is of interest to examine the clinical findings of pocket formation, mobility and suppuration in relation to the high morbidity and tooth mortality figures. Table X and the bar graph (Fig. 11) record these

TABLE X

PERCENTAGE INCIDENCE OF POCKET FORMATION, MOBILITY AND SUPPURATION

Age Group	*Number of Subjects*	*Pocket Formation*			*Tooth Mobility*			*Suppuration*		
		Gen.	*Loc.*	*Tot.%*	*Gen.*	*Loc.*	*Tot.%*	*Gen.*	*Loc.*	*Tot.%*
13-15	121	1	42	43	—	—	—	—	2	2
16-18	94	2	36	38	—	—	—	—	3	3
19-22	117	3	53	56	—	1	1	—	2	2
23-26	86	8	64	72	—	5	5	1	13	14
27-30	79	3	70	73	—	10	10	1	13	14
31-34	73	12	66	78	1	17	18	1	8	10
35-39	102	18	66	84	2	23	25	4	18	22
40-45	100	23	62	85	3	46	49	11	27	38
46-48	129	18	70	88	8	41	49	11	27	38
49-51	104	17	71	88	10	53	63	10	38	47
52-55	79	23	71	94	14	51	65	18	33	51
56-59	64	22	70	92	11	50	61	13	34	47
60 +	39	21	66	87	10	69	79	13	33	46
Total	1187	12	64	76	4	26	30	6	18	24

clinical signs of severe periodontal breakdown.

A high and constantly increasing incidence of pocket formation was revealed in all age groups. Localized pocket formation, confined to one quadrant or isolated teeth or groups of teeth in several quadrants, was predominant, the percentage for all groups being: localized 64 per cent, generalized 12 per cent. The highest percentage, 94 per cent, was registered in the age group 52-55 years.

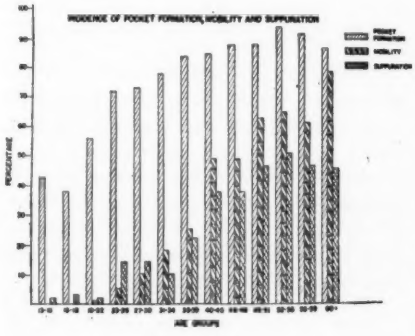

Figure 11.

Tooth mobility rarely occurred before age 27 years. The incidence increased sharply from 25 per cent at age 35-39 years to 49 per cent at age 40-48 years, with a steady increase to 79 per cent at age 60 years. The great majority of subjects exhibited mobility in localized areas, approximately 10 per cent showing generalized tooth mobility from 45 years.

The figures relating to suppuration refer to clinically demonstrable suppuration revealed spontaneously or under digital pressure. Localized suppuration was more common in all age groups. Almost 40 per cent of all sujects showed suppuration at age 40 years and approximately 50 per cent in the older age groups.

Despite the extremely high morbidity and tooth mortality figures and the many obvious signs of serious periodontal disturbance, the questionnaire revealed that only 9 per cent of all subjects were aware of the presence of "pyorrhea" and only 7 per cent had received periodontal treatment.

PREDICTED RESPONSE TO TREATMENT

All clinicians are aware of the difficulties in any attempt to predict the response to local treatment, particularly with a single clinical and roentgenographic examination, due to the almost universal presence of local irritants, the multiplicity of local and systemic differences and the lack of adequate criteria for assessing the relative importance of the various local factors.

With a full appreciation of these difficulties an effort was made to predict the response to treatment in each case, in an attempt to present a more complete appraisal of the periodontal problem in a population group. Cases were tabulated in the simple categories of good, fair and poor. The following criteria were used for determination of the expected response to treatment:

I. Height of Remaining Alveolar Bone: severe bone loss will usually indicate a poor prognosis. Slight bone loss in the presence of unfavorable local factors will indicate good prognosis.

II. Age: In patients with comparable degrees of periodontal destruction and with comparable unfavorable local factors the predicted response to treatment will be better in the older individual.

III. Alterations in Septal Radiodensity—Loss of lamina dura at crest of septum, radiolucent projections into the septum: Slight penetration indicates slow destruction. A slight penetration in the presence of unfavorable local factors indicates good response of alveolar bone. The same degree of penetration in the absence of unfavorable local factors indicates a less satisfactory response. Deep penetration indicates more rapid bone destruction and usually poor response. If associated with severe local factors the predicted response will be more favorable than it would be if local factors were present to a slight or moderate degree.

IV. Distribution of Bone Loss:
If the destructive process is limited to a single tooth or group of teeth the over-all prognosis will be good.

V. Number of Teeth Remaining:
If the number and distribution of remaining teeth are unfavorable for the support of prosthesis, the predicted response will be poor.

Local factors considered were calculus deposition, poor oral hygiene, faulty contacts, faulty restorations and occlusal disharmonies.

These findings are recorded in Table XI. The figures give sharp focus to the fact that early recognition and treatment of the disease could lead to a substantial decrease in the morbidity figures in the older age groups, and that timely diagnosis and adequate treatment at all age levels could result in a marked decrease in tooth mortality from periodontal disease.

SUMMARY AND CONCLUSIONS

As part of a more comprehensive study of the epidemiologic characteristics of periodontal disease, the present report deals with the prevalence and incidence of the disease in the male and female population in Boston. Findings are based upon a careful clinical examination by one investigator and a 20-film intraoral roentgenographic survey including bitewings. For this and for other phases of the survey, study models, kodachrome films, questionnaires, and a medical examination were also used.

In thirteen age groups between 13 and 65 years of age data were recorded relative to prevalence, type and degree of gingival disease and chronic destructive periodontal disease. Particular attention was given to recession and pocket formation, oral hygiene, calculus, type and degree of alveolar bone resorption, tooth mobility, suppuration and tooth mortality.

Some of these findings may be briefly summarized:

TABLE XI

PREDICTED RESPONSE TO TREATMENT (Per Cent)

Age Group	Male			Female			Total		
	Good	*Fair*	*Poor*	*Good*	*Fair*	*Poor*	*Good*	*Fair*	*Poor*
13-15	100	—	—	100	—	—	100	—	—
16-18	100	—	—	100	—	—	100	—	—
19-22	100	—	—	86	14	—	93	7	—
23-26	76	22	2	79	17	4	77	20	3
27-30	61	26	13	54	37	9	57	32	11
31-34	60	27	13	62	28	10	61	27	12
35-39	49	32	19	57	34	9	53	33	14
40-45	48	37	15	48	37	15	48	37	15
46-48	48	42	10	58	26	16	53	34	13
49-51	35	48	17	48	30	22	42	39	19
52-55	52	19	29	60	30	10	56	26	18
56-59	38	50	12	60	34	6	49	42	9
60 +	50	36	14	36	48	16	41	44	15

1. The incidence of gingival disease was extremely high even at an early age, ranging from 80% at age 13-15 years to 95% at age 60, but with a significant reduction to 62% in the late teens and early twenties, particularly marked in the females, and possibly associated with the end of the period of puberty and/or social factors related to a greater emphasis on oral hygiene and awareness of aesthetic values at this age. A sharp rise in both male and female groups was noted at age 23 years and a relatively constant high level of prevalence thereafter.

2. In 10 of the 13 age groups the males showed a higher incidence of gingival disease, with an overall average of 88% and 80% for the males and females respectively. The males also showed a greater degree of severity than the females.

3. There was a consistently high incidence of the marginal type of gingival disease throughout all age groups (average incidence 76%). The diffuse type was encountered with relative infrequency under 40 years of age (average incidence 7%).

4. A study of the regional distribution of gingival disease showed that generalized disturbance was significantly more common than localized disturbance at age 13-15 years. From 16 to 22 years the percentages of generalized and localized cases were approximately equal. From this age there was a comparatively rapid increase with advancing age in the percentage of generalized gingival disease. Generalized disturbance was most prevalent amongst the males, whereas localized disturbance was in most age groups more prevalent in the females. In all age groups the upper posterior regions of the mouth (74%) were more commonly affected than the upper anterior regions (62%). The lower posterior regions (64%) were slightly less affected than the lower anterior regions (66%).

5. In the youngest age group pocket formation with or without recession but no clinical exposure of the root was noted in a significantly higher percentage of the males (46%) than in the females (24%). After 30 years of age a higher percentage of females than males was affected. The total figures decreased with age. Recession with pocket formation and clinical exposure of the root showed a steady increase

in both males and females with increasing age. This condition was not encountered in the males until 19 years of age and in the females until 27 years of age. The incidence was significantly lower in the females in all age groups. Recession with clinical exposure of the root but no pocket formation was encountered in only 5% of both males and females and not before 23 years in the males and 31 years in the females.

6. On the basis of the average number of dental prophylaxes per year and brushings per day the females registered the better performance. The relative importance of these oral hygiene habits as factors in the demonstrated lower incidence of gingival disease in female subjects is a matter for speculation.

7. The incidence of clinically demonstrable calculus showed a marked increase at 19 years of age and a further sharp increase at 31 years, reaching a peak of 91% at 56-59 years. After 30 years of age by far the greater proportion of subjects exhibited both supragingival and subgingival calculus; the average incidence figures for all age groups were: males 39%, females 28%. The correlation between calculus deposition and periodontal disturbance, and the relation between calculus findings and periodontal disturbance in males and females respectively are worthy of note.

8. Chronic destructive periodontal disease was rarely found before 18 years of age, but the incidence increased rapidly from 24% to 69% between the ages of 19 and 26 years, with a steady increase thereafter until 45 years when almost all subjects showed evidence of localized or generalized bone loss. Incidence of bone loss in the males (73%) was slightly higher than in the females (69%). Bone loss without clinical evidence of gingival disturbance was not a common finding (males 7%, females 9%), although the females from 19-35 years showed a greater incidence. There was a consistent increase with age in the incidence and degree of generalized bone loss, until at age 60 all subjects were in the severe generalized category. There was a three-fold increase in the "average resorption figure" between the ages of 22 and 26 years followed by a gradual but steady increase in the older age groups. By age 46-48 years an average of one-fourth, and by 60 years one-third of alveolar bone support was lost. In all seven age groups from the 35-39 year group onwards, areas were found where the supporting alveolar bone was resorbed as far as the root apex. Vertical shearing was slightly more prevalent in the females.

9. The high incidence of pocket formation, tooth mobility and suppuration is important clinical evidence of severe periodontal breakdown. A high and constantly increasing incidence of pocket formation was revealed in all age groups and reached a peak (94%) at age 52-55 years. Abnormal tooth mobility was rarely encountered before 27 years of age but increased sharply from 25% at age 35-39 years to 49% at age 40-48 years, with a steady increase to 79% at age 60 years. Localized or generalized clinically demonstrable suppuration, occurring spontaneously or under digital pressure, was revealed in almost 40% of subjects at age 40 and approximately 50% in the older age groups.

10. The rapid rise in the tooth mortality curve after 35 years of age is significant, and would seem to be related to the morbidity figures and the pattern of chronic destructive periodontal disease which becomes more generalized and more severe in degree by this age. There was a rapid rise in tooth mortality after 40 years of age. By 49-51 years 50% of the teeth were missing. By age 60 years 60% of the teeth had been lost and 26% of the subjects were completely edentulous.

11. Despite the extremely high morbidity and tooth mortality figures only 9% of all subjects examined were aware of the presence of periodontal disease and only 7% reported as having received treatment for this condition. These findings point to an urgent need for greatly increased emphasis on periodontology in the

dental schools and in dental practice. This need is underlined by the less discouraging findings in relation to the predicted response to treatment.

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Therapeutic Nutrition for the Periodontal Patient

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GINGIVAL and periodontal health is, in part, dependent on acquiring, utilizing and absorbing the nutrients of a balanced diet. What, why and how to prescribe this balanced diet to meet the individual needs appears to be the desire of the profession.[1]

For many medical problems (diabetes, cardiac disease, gall bladder dysfunction) a specific nutritional relationship has been established. This cause and effect relationship between specific nutrients and periodontal disease has not been adequately shown to date. However, the periodontist is intimately concerned with epithelial keratinization, vascular integrity, osteogenesis, wound healing and tissue repair which are all physiological processes that are directly related to nutritional biochemistry.

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It appears that there is a need for the application of basic principles of therapeutic nutrition in periodontal management. A brief comment on the nature of these basic principles, a method of nutritional appraisal and some suggestion on prescription of nutritional supplements will be the substance of the foregoing discussion.

BASIC CONCEPTS

Nutrition connotes a study of biochemical changes of cells and tissues through ingestion, and utilization of the chemical nutrients of foodstuffs. Without an appreciation of the pathogenesis of a nutritional deficiency, or the importance of protein in antibody formation, or the realization that vitamins are interrelated and interdependent, a rational for proper treatment cannot be attained.

A deficiency results whenever amounts adequate for the normal functioning of tissue are not provided. This may be caused by a primary inadequacy which is purely a dietary deficiency or a secondary conditioned inadequacy which deals with the ability of the body to ingest, absorb, and utilize the ingested foodstuff. However, a nutritional deficiency will not take place until sufficient time has elapsed to deplete the body of its nutrient reserve. Once this has come about then biochemical differences, functional changes and anatomical lesions will take place.[2] These latter three events are detected by laboratory and clinical examinations and are basic information for the assessment of one's nutritional status.

Proteins are important because the ability of the body to combat disease is dependent on the presence of antibodies. Since antibodies are protein there seems to be no other mode of antibody synthesis but from the amino acids of dietary protein. According to Carron[3] one portion of the serum protein (the globulin) carries with it the various antibodies against infectious agents. Therefore, protein undernutrition is associated with susceptibility to bacterial invasion.[4]

Protein deficiency has been definitely shown to be a factor in postoperative wound healing[5] as well as periodontal disturbance[6] in experimental animals. On the other hand, high protein diets adequate also with respect to riboflavin, Vitamin C and Vitamin A shorten the lag period in repair of wounds.[7] To date in man this relationship has not been definitely shown but is empirically accepted.

Vitamins are accessory food substances. They are components of enzyme systems which are essential for cell and tissue health. For example, a deficiency of Vitamin C could produce a failure of osteoblasts to form osteoid, the organic matrix of bone, as well as be responsible for the failure of collagen formation from immature fibroblasts.

Another characteristic of vitamins is that they are interdependent and interrelated. The lack of vitamin C will affect the metabolism of vitamin B. Therefore they are invariably prescribed as multivitamin preparations.[8] It has been recently demonstrated that certain oral lesions which were thought to be representative of a specific vitamin deficiency may actually be produced by lack of any one of several vitamins. For example, atrophic lingual glossitis or cheilosis may respond to iron, riboflavin, or pyridoxine or a combination of all three.[9]

The rapidity of onset and duration of a vitamin deficiency is an important consideration from a therapeutic standpoint. Acute deficiencies respond quickly to therapeutic doses. On the other hand, deficiencies insidious in onset and chronic in duration have no need of massive doses but merely require smaller maintenance ones over continuous periods.

These are only a few of the basic principles that guide one toward proper nutritional assessment as well as nutritional therapeutics.

NUTRITIONAL ASSESSMENT

The first step in evaluating a patient's nutritional status is to obtain a dietary history. This is done by requesting that a diary be kept of foodstuffs eaten for seven consecutive days. Transposing this information to a single qualitative evaluation chart as described in Figure 1 might reveal a primary nutritional deficiency. In this type of chart information on not only the quality of the diet is elicited but also its physical nature. The non-detergent foods are generally classed as animal protein type or energy type. The detergent ones are either fruits or leafy, green and yellow vegetables. In general these are the four types of food that should be eaten at each meal each day for a balanced diet.

1. Animal protein foods—milk, meat, eggs, fish, poultry, to furnish amino acids plus vitamins and minerals.

FIGURE 1

Qualitative Evaluation of Dietary Intake Chart

	Class 1				Class 2				Class 3				Class 4			
	Animal Protein Foods (Meat, Eggs, Fish, Poultry, and Milk.)				Energy Foods (Cereals, Breads, Fats, & Sugars.)				Green, Leafy & Yellow Vegetables				Tomatoes, Melons, Citrus & Other Raw Fruits.			
	B	L	D	E	B	L	D	E	B	L	D	E	B	L	D	E
Example:	√	√	√	O	√	√	X	O		O	X	O	X	√	O	O
Monday																
Tuesday																
Wednesday																
Thursday																
Friday																
Saturday																
Sunday																
	NON-DETERGENT								DETERGENT							

NOTE: These four classes of foodstuffs should be eaten consecutively. For the purpose use √ if eaten in ideal order, X if not ideal, and O if absent.
For example the following intake would be charted as above:
B—Orange, Egg, Toast and Coffee.
L—Ham Sandwich, Milk and Fruit.
D—Salad, Meat, Cake, and Tea.

B—Breakfast L—Lunch D—Dinner E—Extras

2. Energy foods such as cereals, fats, sugars to provide, in the main, for the caloric needs as well as some vitamins and minerals.

3. Green, leafy and yellow vegetables to furnish minerals, vitamins, and fibrous bulk.

4. Citrus and other fruits, tomatoes to furnish vitamin C, other vitamins, minerals and general detergency to the diet.

By using √ mark in the appropriate blocks when the food is eaten in the ideal order from a detergent standpoint, x when the food is eaten but not in the ideal sequence, o when it is omitted from the meal, one can readily point out gross qualitative omissions and errors. Ideally of course, all blocks for breakfast, lunch, and dinner should have a √ mark.

The second step is to determine through a simple medical history whether there are systemic factors present which influence the utilization and absorption of the nutrients.[10] Some of the most common conditioning factors are listed in Table 1.

The third step is to look for some physical signs of nutritional deficiency. These are:

1. General appearance of debility and pallor.

2. The skin may be dry, rough and pigmented with an excess of blackheads and whiteheads about the nose and mouth. There may be excess of subcutaneous fat, loss of elasticity and wrinkling on stroking. Symmetrical dermatitis, ulcerations and exfoliations on the backs of the hands and in-steps of the feet sometimes may be found.

3. Poor posture and general muscle incoherence should be observed.

4. There may also be a history of photophobia or night blindness.

5. The angles of the mouth may show dryness and fissuring. The dorsum of the tongue may be smooth, glistening, beefy red or magenta in color, as a result of atrophy of the papillae. The gingiva may be hemorrhagic and edematous.

6. The extremities reveal muscular tenderness, weakness, peripheral neuritis, wrist drop, or ankle drop.

The fourth step is the use of pertinent laboratory examination of the blood and urine. Iron is estimated by first determining the level of hemoglobin in the blood, the hematocrit, and the red cell count. From these data the mean corpuscular volume and the mean corpuscular hemoglobin are determined. Ascorbic acid levels in plasma, whole blood and white cells as well as "load tests" are used to determine the adequacy of vitamin C. The blood lactate-pyruvate relationship has been used in evaluating certain vitamin B deficiencies. Chemical tests for vitamin A nutrition include determination of the concentration of vitamin A and carotene in the blood. The nutritional status of vitamin D and of calcium is appraised by determining the level of calcium in the blood and the amount of alkaline phosphatase in the serum.

These four steps—dietary history, medical history, clinical and laboratory examination—make up the factors essential for an overall assessment of a patient's nutriture. From a practical standpoint, it will be found that laboratory procedures are seldom used. However, all the other three factors are invariably taken into consideration.

THERAPEUTIC NUTRITIONAL PROCEDURES

If there are nutritional abnormalities, the assessment of the periodontal patient will resolve itself into these basic considerations.

1. The conditioning factors preclude a proper utilization of the ingested foodstuffs.

2. The intake is inadequate and unbalanced.

3. The physical nature of the diet does not contribute to promoting good oral physiotherapy.

4. Supplementation with nutritional preparations is indicated.

The systemic conditioning factors are not within the scope of the periodontist to treat, but they are his responsibility to recognize and channel in the proper direction.

On the other hand, the alteration and correction of dietary factors to meet the needs of maintaining and restoring gingival health are his responsibility. Dietotherapy should be based on including daily the so-called "basic 7"—milk, leafy, green and yellow vegetables, legumes, fruits, breads, meats, and fats. For practical periodontal purposes we have regrouped them into two main types, detergent and non-detergent, and have further subdivided them into (1) animal protein, (2) energy type foods, (3) leafy, green and yellow vegetables, and (4) fruits. All these foods should be eaten at each meal. (See Figure 1.)

The physical nature of the diet is im-

TABLE I

CONDITIONING FACTORS

Gastroenteritis
Peptic ulcer
Diarrhea disease
Gallbladder disease
Liver disease
Congestive heart failure
Food allergy
Operations
Alcoholism
Edentulism
Pregnancy
Lactation
Diabetes
Hypothyroidism
Hyperthyroidism
Anorexia
Rheumatic fever
Malignancy
Tuberculosis
Use of thyroid extract
Chronic indigestion
Measles
Pertussis
Scarlet fever

portant from a self-cleansing and tissue stimulating standpoint. For these reasons we recommend that the chewy cellulose type foods such as raw vegetables and fruits be used at the end of each meal. The qualitative evaluation chart previously discussed incorporates this feature and portrays rather graphically obvious errors in the patient's dietary pattern.

The fourth consideration in therapeutical nutrition procedures is prescription of nutritional supplements. In periodontal disease and surgery, maintaining adequate vitamin and protein levels is desirable. During acute or active disease, the water soluble vitamins B and C are most likely to be inadequate due to two conditioning factors. There is an inability to ingest natural, raw, chewy fruits and vegetables due to pain. There is an increased requirement as well as an inability to absorb and utilize efficiently the available vitamins due to local vascular congestion and inflammation.

In the basic concepts, two points concerning vitamins have been made, namely, (a) their interdependence and (b) that the acuteness or chronicity of the condition influences the amount to be prescribed. Therefore, in prescribing vitamin supplement two rules should be followed. First, multiple vitamin tablets or capsules of B complex and C should be prescribed rather than individual vitamin preparations. Secondly, in the acute phases where the duration of the disease is short and its nature severe, a large or therapeutic dose is indicated for rapid recovery. This usually is five times the N.R.C. recommendation. The following is the formula that is advisable:

Thiamine	10 mg.
Riboflavin	10 mg.
Niacinamide	100 mg.
Calcium Pantothenate	20 mg.
Pyridoxine HCl	2 mg.
Folic acid	1.5 mg.
Ascorbic acid	300 mg.
Vitamin B_{12}	4 mcg.

If a diagnosis of frank scurvy is made then additional vitamin C up to 1 gram is prescribed.

For chronic insidious types of periodontal disease where there are doubts about the adequacy of current or previous diets then maintenance doses of the following is advisable:

Thiamine HCl	2 mg.
Riboflavin	2 mg.
Nicotinamide	20 mg.
Ascorbic acid	50 mg.
Calcium Pantothenate	5 mg.
Pyridoxine	0.5 mg.
Folic acid	0.25 mg.
Vitamin B_{12}	2 mcg.

In addition to vitamin supplementation, it has been shown that during periods of catabolic stress, increasing protein intake might have a curative effect and more rapidly restore tissues to normal. Eggs and skimmed milk powder are two excellent sources of quality proteins with limited bulk, bland flavor, and compared to flesh proteins are economical. The following high protein eggnog is an excellent protein supplement, because it provides 19 gms. per cup which meets one-fourth the daily requirement.

4 eggs
½ tablespoons sugar
1 quart whole milk
1 cup skim milk powder

Beat eggs with sugar. Add milk and mix. Sprinkle milk powder over milk-egg mixture and beat until smooth. Flavor with vanilla. Chill thoroughly before serving.

CONCLUSION AND SUMMARY

Therapeutic nutrition can be and should be used as an adjunct to periodontal management by simply adhering to five simple principles. (1) Apply some of the basic underlying concepts to a rational of nutritional management. (2) Do more than a simple diet evaluation when assessing the patient's nutriture by including a brief medical history and clinical examination. (3) Prescribe a diet based on a modification

of a normal balanced diet. (4) Prescribe detergent foods at the end of each meal as an additional means of oral physiotherapy. (5) Use nutritional supplements particularly vitamin preparations based on the quality and duration of the disease.

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Sodium Silicofluoride as a Desensitizing Agent

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SENSITIVITY of exposed cervical cementum is a common complaint in adults. The pain may vary in degree and intensity from a slight sensitivity to excruciating pain, and one or a number of teeth may be involved. Merritt[1] classifies sensitivity into two types: (1) surface irritation, (2) thermal shock. This sensitivity is excited by touch, sweets, condiments, citrus fruit juices, and heat or cold. The two types of sensitivity often do not occur together.

Exposure of the cemento-enamel junction may be physiologic for patients in the older age bracket. In other patients, it may be brought about by toothbrush abrasion or abrasive dentifrices. The occurrence also may be noted following surgical procedures, such as the subgingival curettage and gingivectomy in periodontal surgery, and following extraction of teeth where bone is removed, leaving exposed cementum on adjoining teeth. Repeated injudicious scaling of root surfaces, and retention of foods with acid formation in cases of poor oral hygiene are other factors to be considered.

A quick glance at the histology of the cementum relieves any doubt as to why this area may become so sensitive. Orban[2] describes the cemento-enamel junction as a union which takes place in the following manner: in 60% of the cases the cementum overlaps the enamel; in 30% there is an end to end joint, and in 10% there is a void where the dentin is exposed. The cementum is thinnest at this junction varying in thickness from 20 to 50 microns as compared to 150 to 200 microns at the apex. This extremely thin covering of cementum, even when intact and histologically normal, offers a minimum of protection against irritation and thermal shock. However, it is frequently removed

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by abrasion, and almost always by curettage.[3]

In the past, many methods of treatment have been described. The main objective of the drugs used was to precipitate albumin, thus causing a seal of the dentinal tubuli and thereby decreasing sensitivity. For surface irritation 40% formalin, 40% zinc chloride, and 40% silver nitrate (posterior areas) have all been used with varying degrees of success. In the treatment of thermal shock cavity varnishes, thin mixes of crown and bridge cement, zinc oxide-eugenol packs, and Gottlieb's solution have been mentioned.

In the periodontia clinic at Baylor, for the past twelve months, we have used sodium silicofluoride (Na_2 Si F_6) in 0.9% solution to treat all types of cervical sensitivity. After cleaning and drying the sensitive teeth, the solution is applied with cotton applicators, keeping the treated areas glistening but not dripping wet, for a period of five minutes. No attention is given the gingivae, since the 0.9% aqueous solution is not caustic or irritating to the tissue. If one application is not sufficient it must be repeated weekly for a total of three treatments. Best results are obtained by applying the solution to the affected areas immediately following prophylaxis, as this assures a clean surface. No amount of overemphasis can be placed on having the teeth to be treated thoroughly dry and isolated by cotton rolls.

A fresh solution should be made weekly and stored in plastic containers, but never in a glass bottle due to a chemical reaction between the fluoride and glass.

The 0.9% solution may be made quickly by weighing 4.08 grains of sodium silicofluoride and dissolving in 30 cc of distilled water. The mixing of future solutions is simplified by preparing and labeling a large number of packets of powder ready for instant use by adding an individual packet previously weighed to the required amount of distilled water.

The only precaution necessary in the use of this drug is to remember it has toxic properties and as little as .05 gm. may cause prolonged nausea, slowing of pulse and lowering of blood pressure. Avoiding an excess in application, thorough rinsing of the mouth immediately after removing the cotton rolls, and the use of a saliva ejector to prevent swallowing until the mouth can be rinsed are the procedures to be followed carefully in order to insure complete safety.

Shaw[4] claims the action of topical fluoride brings about a slight increase in the fluoride content of the enamel surface and reduces the acid solubility of enamel by forming calcium fluoride.

From clinical results obtained in the use of sodium silicofluoride as a desensitizing agent for exposed cementum, we find that the tooth surface seems to be affected less by acids, by other surface irritants, and by thermal shock. In fifty-five treated patients using this solution, in all types of cervical sensitivity from varying stimuli, and in patients complaining of sensitivity, pain, and discomfort in one tooth or in multiple teeth, the following results were registered: Complete success was achieved in thirty-seven patients; partial success in thirteen patients, each of whom described the sensitivity as modified but still noticeable; and in five patients poor results were obtained. It is interesting to note in thirty-five patients only one treatment was necessary; in nine patients two applications were applied; and in eleven patients three applications were applied. A fourth treatment was tried in two of the five patients with poor results and no improvement was noticed.

DISCUSSION

This treatment offers a simple method of desensitization of exposed cementum with a single non-caustic, non-irritating drug. This is advantageous in that the gingivae are not cauterized, resulting in tissue slough and further exposure of the ce-

mentum. The technique of application is simple, requires no burnishing, no precipitation of the drug, and no more precaution in its use than any other agent used for desensitization.

SUMMARY

1. Sodium silicofluoride in 0.9% aqueous solution has been employed as a desensitizing agent for sensitive teeth, with better results than any other drug or drugs previously used for the purpose.

2. No known single method of treatment for this sensitivity is 100% effective.

3. Areas of exposed cementum which have previously been treated should be given a single treatment immediately following routine prophylaxis.

4. The permanency of protection in successfully treated patients will last from prophylaxis to prophylaxis provided there are no clasps or similar mechanical areas of abrasion.

5. Patients who are not benefited after three appointments will not be aided by additional applications.

6. Sodium silicofluoride in 0.9% solution does not irritate the pulp or discolor the tooth.

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IN MEMORIAM

Clayton H. Gracey, D.D.S.
1888-1955

Members of the American Academy of Periodontology, and all dentists who knew him, were saddened by the death on April 2, 1955, of Dr. Clayton H. Gracey, well known periodontist of Birmingham, Mich.

Dr. Gracey was born June 13, 1888, in Comber, Ontario, and was graduated from the University of Michigan and the Detroit Dental College. For many years he practiced in Detroit, specializing in periodontology. In 1926 he moved his office to Birmingham, Michigan, and practiced there until he retired in 1952.

Dr. Gracey was a pioneer in the field of periodontology and made many fine contributions which helped advance science in this field. He was always a professional gentleman, was active in the American Academy of Periodontology and other dental organizations and was widely known for his scientific approach to dental problems.

The More Important Advances in Periodontal Research and Their Clinical Implications

by DONALD A. KERR, D.D.S., M.S., *Ann Arbor, Michigan*

AT the meeting of the American Academy of Periodontology in Cleveland last year Harold Ray, as chairman of the program committee, gave a very comprehensive report of the research in progress in the various schools. The report stated, "Our study reveals that there is insufficient correlation between the clinical projects and the biologic. While all of the studies being conducted have a very definite relation to the problems in Periodontics, the over all picture does not present an orderly continuity. The clinical group of studies now being conducted can be most pointedly criticized. This is a heterogeneous group of projects having no particular direction. They all pertain to our problem, of course, but have been dreamed up individually, stimulated by a variety of interests. One can see clearly the enthusiastic influence of the drug houses in evaluating their products, often resulting in needless repetition. There are too the hobby horses ridden by enthusiasts for certain principles already evaluated."

The assigned title of this paper was "The More Important Advances in Periodontal Research and Their Clinical Implications." This title would direct the writer to evaluate all the recently reported research and weigh the importance of each project and then relate it to clinical practice. Time did not permit such a survey, nor does the time allotted here permit such a report. Also, the writer lacks confidence in his ability to make such a report. I would, therefore, like to speak on the relationship of research to clinical practice.

In studying the 1952-53 report of the research committee, one finds that 28 schools reported 100 projects in progress. Forty-four of the investigations were biologic in character, 30 were bio-clinical, and 26 were strictly clinical problems. These figures give us some idea of the relationship of research to clinical practice, as 56 of the problems indicated some degree of clinical application. This points out, however, only a relationship to clinical practice and does not indicate the impact on clinical practice. Many of the investigations, although clinical in nature, may be without significance when completed especially those having to do with the testing of various commercial products.

In reviewing the recent periodontal literature, the research reports fall into certain rather well defined categories which can be discussed as a group.

Biological problems comprise a large part of the presently reported periodontal research. Typical examples of this type of research are illustrated in the following titles: "The Effect of Various Levels of Dietary Protein on the Periodontal Tissues of Young Rats," "Effect of Dilantin Sodium on Rat Gingiva," "Mitchell Index of Gingival Changes in Alloxan-Diabetic Hamsters," and "Mitotic Activity in Periodontium of Rat Molars." These are only a few examples picked at random to illustrate the type of biologic problem being investigated. This type of problem serves the very desirable purpose of training young investigators in research methodology and its report serves as a partial requirement for an advanced degree. Biologic research gives specific information regarding a selected physiological process which is usually only a small part of a more complex general physiological problem. Basic biologic research has many facets which must be fitted together to complete a complex picture. The solution of one problem indicates that numerous others of a more difficult nature also need investigation before the clinical

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significance of the first problem will be evident.

The presence in the literature of reports on numerous unrelated biologic subjects indicates the need of some form of correlation between many investigators if biologic research is to have clinical implications. Atomic research has pointed to the advisability of having such a correlated effort in basic research. It would, therefore, be desirable to have all the research in progress reported each year to the research committee and published in the *Journal*. This would permit numerous investigators to correlate their activities, and it would also prevent duplication of effort.

The reporting of the results of biologic research frequently infers that the findings are basic biologic principles which can be translated into clinical periodontal practice. The inference is often times so strong that the clinician accepts it as fact and applies it to clinical practice without taking into consideration that the morphology, histology, physiology, and metabolism of small laboratory animals are not comparable to man. In many of the dietary investigations laboratory animals are deprived of specific food factors and inference is then made that the observed findings are indicative of a deficiency of the specific food without the realization or rationalization that deprivation and deficiency are not synonymous and do not produce the same results. None the less, the evidence that a white rat fed on a protein deficient diet develops periodontal disease is frequently accepted as evidence that a man deficient, but not deprived, of protein will develop periodontal disease. This and similar evidence frequently affects clinical periodontal practice, because the inference is so strong that it is true for man and also because the clinician fails to exercise good scientific judgment in evaluating the research report. These examples serve to point out the possible errors which arise from incorrect interpolation of findings on incomparable biologic mechanisms.

The problems of a similar type are illustrated by the titles, "Effect of Cortisone Upon the Periodontium of White Mice"[2] and "The Effect of Estrogen on the Gingiva and Alveolar Bone of Molars in Rats and Mice."[3] This type of investigation is designed to demonstrate the periodontal changes which result from altered hormonal balance, although the quantity of substance administered is excessive and does not duplicate the conditions which are of natural occurrence. That these findings cannot be applied to man is well illustrated by the studies of the effect of estrogen in laboratory animals in which it is found that the effect of estrogen varies not only in species, but also in different stages of development and in different areas of the skeleton in the same species.[4]

Such investigations are usually designed to study the systemic factors in periodontal disease and show that by alteration of the normal physiology of the animal periodontal changes can be produced. If there was evidence that the findings were valid for man, they would favorably affect clinical practice, but their frequent acceptance through inference and their placement into clinical practice as a replacement for more arduous time tested methods of practice is to be condemned.

Another form of animal research involves procedures carried out on the periodontal tissues of various types of laboratory animals. This type of investigation is illustrated in titles such as, "Mitotic Activity of Fibroblasts After Damage to the Periodontal Membrane of Rat Molars"[5] or "The Influence of Injury to the Periodontal Membrane on Spread of Gingival Inflammation."[6] Although not stated in the title, this investigation was on rats. The elimination of the designation "on rats" gives the inference that this is true of any periodontal membrane. Investigations of this type are frequently based on the assumption that the reaction of rat and man are comparable and does not take into consideration the difference in morphology of the teeth and periodontal membrane of the

two animals. Findings from this type of investigation may give information regarding the specific reaction of a specific tissue to a particular type of stress. The value from such research may be made more applicable to clinical practice if the selected laboratory animal closely simulates man. That is, a monkey would be more comparable than a rat.

Still another category of research frequently investigated is that carried out on the human periodontium. Examples of this type are: "Structures of Epithelial Attachment Revealed by Phase Control Microscopy,"[7] "Reattachment of Human Periodontal Tissues Following Surgical Detachment,"[8] "Removal of Pocket and Attachment Epithelium in Humans,"[9] and "The Gingival Sulcus and the Periodontal Pocket Following the Scaling of Teeth."[10] Investigations of this type carefully pursued and adequately controlled may yield information of great value in clinical practice. The results of such an investigation may alter or influence remarkably certain clinical procedures and serve to disprove incorrect clinical impressions which now are a part of clinical practice. This type of investigation is difficult to pursue without the presence of many variables, any one of which may alter the results and incorrectly affect clinical practice. Whole hearted acceptance without critical evaluation is to be avoided.

Studies regarding the prevalence of periodontal disease such as "Gingival and Periodontal Disease in Children,"[11] "Prevalence and Incidence of Alveolar Bone Disease in Men,"[12] and "Occurrence of Gingivitis in Suburban Chicago School Children,"[13] when carefully made on large population groups can give valuable information which has direct clinical application. Findings from research of this type are especially applicable to preventive periodontia. Much can be learned regarding the age of onset of periodontal disease, the characteristics of incipient periodontal disease, and the frequency of occurrence. Such information could be a potent factor in the diagnosis and management of periodontal disease.

Titles such as "Some Investigations on the Cleansing Effect of Nylon and Bristle Brushes,"[14] "Calculus Reduction with a Mucinase Dentifrice,"[15] and "Hydrocortone in the Treatment of Gingival Disease," indicate another field of research which is clinical in nature. This type of research is necessary because of the frequency with which new brushes and dentifrices and other aids to periodontal therapy are being placed on the market. Even though these are commercial products, often highly advertised, they should be studied to determine whether they have therapeutic value. Although the dental profession fully realizes that it is not the brush but how it is used that is important, we should also know which, if any, is most efficient. Dentifrice manufacturers are continually developing new products to which are added various enzymes, wetting agents, and antibacterial agents. Each of these should be carefully evaluated regarding their therapeutic value. This is frequently difficult because of the inability to control the investigation and evaluate a single factor. Too enthusiastic acceptance of results of this type of investigation frequently gives incorrect clinical evidence as has been demonstrated in the control of caries.

Reports of specific disease processes as is illustrated by the titles "Psoriasis Gingivae"[16] and "Gingival Manifestations of Lupus Erythematosis"[17] indicate investigations of specific disease processes which may affect the gingivae. Too frequently these are case reports of clinical findings without histological study to verify the diagnosis. However when these case reports are based on good clinical observation, adequate histories, and complete histological study they improve our clinical practice through more accurate diagnosis and more positive therapy.

The categories of research mentioned do not in any way attempt to cover the entire field of periodontal research but were selected to serve as examples of specific types

of problems being investigated. It can be observed that much of the research being carried out is basic in nature and has only a limited effect on periodontal practice, and that the effect which is exerted on clinical practice is on some occasions an undesirable one. This does not imply that because biologic research does not have direct clinical application that it is of no value. However, I do wish to caution against the acceptance of findings in laboratory animals as fact directly applicable to periodontal problems in man.

Evaluation of recent periodontal research indicates the need for greater correlation of basic research problems. The elimination of inferences that findings on laboratory animals can be translated to man. The cessation of the practice of selecting research projects primarily for the purpose of obtaining grants in aid to acquire personnel and laboratory equipment. It would also be desirable to restrict reporting in the literature of the less important problems investigated as partial requirement for a Master's degree.

In contrast it would be desirable to have co-operative activity between research workers so that several investigators in different locations would each investigate a particular facet of a single problem which would give more valuable results—some of which could influence the treatment of periodontal disease.

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DR. HOLLERS HONORED

The many friends of Dr. James P. Hollers of San Antonio will be pleased to learn that he was recently promoted to the rank of Brigadier General in the Air Force.

DISCUSSION OF DR. KERR'S PAPER

by ROBERT KESEL, D.D.S., M.S., *Chicago, Illinois*

Dr. Ray and Dr. Kerr both have suggested closer coordination of research in Periodontics in order to raise standards, to develop more orderly continuity in the pursuit of research and to avoid duplication of effort. The surveys that they have made of research in progress and of titles and content of research reports in our literature reveal that the current pattern of attack on periodontal problems is heterogeneous. A better organized effort might be more rapidly productive. However, I do not believe that the condition of research in periodontics differs qualitatively from that of research in other fields, and I wonder if it would be desirable to fetter investigators by laying out a pattern and requesting that it be followed. Investigations are born of ideas and there is an essential spontaneity about them. Research must be pursued with diligence and enthusiasm, and if an individual does not feel completely free in the exploration of his thinking; if there is a pattern which he must follow or a suggested goal toward which he must point his interest, his productivity may be stifled. Good investigators are individualists and the antithesis of conformists. It has been stated that the success of atomic research points to the advisability of correlated effort in basic research. It is my impression that atomic research is notoriously handcuffed, regimented and gagged, but necessarily so for security reasons.

There seems to be a desire to avoid duplication in research, and I realize that there is some duplication of effort and overlapping of ideas. However, duplication is not all bad; in fact, it is essential, because the results of any investigation must be verified independently before they can be accepted more than tentatively.

Actually, complete repetition of many investigations would be desirable. Unfortunately, even when investigators set out conscientiously to duplicate a given piece of biological or clinical work, they find their task very difficult due to variations in the composition and behavior of the human or animal material they must study. Under prevailing conditions, most researchers also introduce their own variations, and discover when they finish their duplicate study that the variations in material plus their own deviations from the design of the original study have defeated their attempt at duplication.

Dr. Kerr and Dr. Ray appear to criticize research, especially clinical research, on so-called commercial preparations. We must remember that manufacturers support extensive and expensive research programs manned by well-trained individuals in their own and in other organizations. It is only logical to believe that they will develop a number of potentially beneficial products, but obviously such products cannot be accepted for general use until they have been studied adequately by other unbiased groups. I don't believe we should disparage carefully done and properly controlled clinical research with such preparations. On the contrary, I wonder if we shouldn't encourage such investigations so that we may get necessary information from reliable sources more rapidly.

It is apparent that authors should frame the titles of their research reports more carefully. The title should disclose the area of study and the kind of subjects used. If animals are used their type should be stated. Our literature is becoming more and more voluminous, and progress reports or problem reports that are developed in fulfillment of the requirements for a Master's Degree should be carefully screened by editors.

I believe that we are on the verge of rapidly expanding frontiers in our knowledge of periodontal diseases and their prevention. Advances will be stimulated by

our newer research tools such as histochemistry, radioactive isotopes, and the electron microscope. I think these new tools will help us to solve a number of problems including calculus deposition, bone metabolism, the detachment of the periodontal fibers and the downgrowth of the epithelial attachment. As a result, we may look forward to the preservation of more teeth in coming generations.

I concur in the suggestion that an active committee on research in the Academy should obtain information about current investigations in Periodontics and related science in this country and also in foreign lands, and should make this information available to the Academy, to the Editor of our Journal, and to the dental profession. This committee should take into account one of the functions of the recently established Council on Dental Research of the American Dental Association, which I understand is to survey the entire field of dental research in the United States and to evaluate research facilities. Perhaps a mutually helpful working arrangement with the Council on Dental Research could be developed by our research committee. The Academy and its Journal could serve as a clearing house for research information, but I doubt that any attempt should be made to direct or regiment research.

An Evaluation of the Peripheral Giant Cell Tumor

by RICHARD L. PHILLIPS, D.D.S.,* AND WILLIAM G. SHAFER, D.D.S., M.S.,*
Indianapolis, Indiana

LITERATURE on the subject of peripheral giant cell tumors dates back at least to 1818 when Cooper[1] described such lesions. While many of the early writers on this subject felt this lesion to be malignant on the basis of its tendency to recur if incompletely removed, others believed it to be benign. Virchow[2] was of the opinion that the lesion could become malignant or had the tendency to recur with malignant possibilities. Paget[3] thought the lesion benign but had a tendency to recur if not completely removed. Sugar[4] states that the lesion "cannot be classified as a malignant tumor because its growth is not of an infiltrative character, it does not develop metastases, and its histologic picture lacks signs of malignancy such as the shrinking of the nuclear chromatin substance, pyknosis, and atypical multipolar, hypo- and hyperchromatic mitosis." Stones[5] and Geschickter and Copeland[6] support the theory that the lesion is benign. It is the general opinion today that the tumor is benign but has the tendency to recur if incompletely removed.

This lesion has been named "myeloid sarcoma" and "myeloid epulis," neither of which is applicable because there is no evidence of metastasis as the term "sarcoma" designates, nor does the tumor have any connection with the bone marrow as is indicated by the use of the term "myeloid."[5] Stewart[7] (1922) suggested the term "osteoclast sarcoma" on the basis of his conclusion that the giant cells were osteoclasts. This expression possibly was the origin of "osteoclastoma" used by some writers today. Bernier and Cahn[8] found no evidence that the giant cells are produced to remove bone even though they bear a morphologic similarity to osteoclasts, and that the term "osteoclastoma" seems inappropriate. The terms "giant cell epulis" and "peripheral giant cell tumor" are used commonly today along with "peripheral

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giant cell reparative granuloma" which Bernier and Cahn[8] have suggested, in that it is similar to the lesion described by Jaffe[9] as "giant cell reparative granuloma of the jaw bones."

It is appropriate to say at this point that there are two schools of thought as to the nature of this lesion. One group believes the lesion to be a true neoplasm whereas the other group believes it to be of an inflammatory nature.[10] A recent paper by Bernier and Cahn[8] is concerned with establishing the true nature of this lesion. They suggest that the relationship of Jaffe's[9] "giant cell reparative granuloma of the jaw bones" and their "peripheral giant cell reparative granuloma" to trauma is an important factor in distinguishing these lesions from the true giant cell tumor of bone.

The origin of the giant cells is a confused issue of which there are many theories and none have been definitely established as correct. Ritter[11] and Mezrow[12] believe the giant cells originate from the endothelial cells of the capillaries. This is a good possibility because histologic sections from peripheral giant cell tumors often show giant cells within the vascular channels. Rywkind[13] (1927) believes the tumor to be a reactive growth of granulation tissue. Sugar[4] (1948) suggests that the giant cells are the result of fusion of connective tissue cells or a division of the nuclei of connective tissue cells without division of the cytoplasm. Bernier and Cahn[8] and Jaffe[9] believe the lesion to be a local reparative reaction. Geschickter and Copeland[6] suggest that, since the lesion occurs in the region of the deciduous teeth, it may be associated with the shedding process. They state that "about the roots of the deciduous teeth, therefore, there is a periosteum which is normally endowed with the power of osteoclasia. This osteoclastic hyperplasia is a normal occurrence after the age of five and is nature's provision for shedding the deciduous teeth. The process is analogous to the osteoclastic hyperplasia that occurs at the ends of the long bones and in the cartilagenous bone. The deciduous teeth therefore are in part like cartilagenous bone in that both are temporary bone structures and the pathologic process of giant cell tumor arises in connection with either, in one case giving origin to epulis and in the other to the typical giant cell tumor of long bones."

Stones[5] agrees with this concept but further states that in cases of peripheral giant cell tumor posterior to the bicuspid region, the giant cells may possibly arise from the undifferentiated mesenchymal cells of the periodontal membrane "as it is easy experimentally to stimulate the production of osteoclasts in this tissue."

CASE REPORTS

Sixteen cases were available for study from the files of the Department of Oral Pathology, Indiana University School of Dentistry and cover a period from November, 1949 to December, 1954. During this interval, 2,228 biopsy specimens were submitted; the peripheral giant cell tumor thus represents approximately 0.7 per cent of all lesions processed.

Case #1. A thirteen year old Negro boy presented with a pedunculated mass 1x5x2 cm. of 6-8 weeks' duration, labial to the lower left central and lateral incisor. He gave a history of being struck in the mouth in this region while playing football. Roentgenographic examination showed no involvement of bone.

Case #2. An eleven year old Negro boy presented with a small growth on the palate lingual to the upper right central incisor. The mass was a small pedunculated, discoid tumor measuring approximately 2 cm. in diameter and similar in color to the mucous membrane of the mouth with attachment at the gingiva and periodontal membrane. Roentgenographic examination showed no unusual condition of the bone underlying the mass.

Case #3. A seven year old white girl presented with a painless growth about 15 mm. in diameter and of several months' duration on the edentulous ridge of the lower right deciduous molar area.

Case #4. A thirty year old white female presented with a tumor of the left mandibular cuspid region on the crest of the edentulous ridge. At the time the tumor was first noted, the patient was in the eighth month of pregnancy. The mass had been present for nine months at the time of excision.

TABLE 1
Summary of 16 Cases of Peripheral Giant Cell Tumor

Case #	*Age*	*Sex*	*Race*	*X-ray Evidence of Bone Involvement*	*Location*	*Duration*
1	13	M	C	No	⌈1 region	6-8 weeks
2	11	M	C	No	Palate posterior to 1 ⌋	4 weeks
3	30	F	W	—	3 ⌋ region on edent. ridge	9 months
4	7	F	W	—	⌈5 region on edent. ridge	several months
5	15	F	W	No	Labial to 2 ⌋	4 weeks
6	—	M	—	—	Labial to 3 ⌋ socket and adjacent gingiva	1 year
7	16	F	W	—	Between 1 ⌋ 2	8 weeks
8	39	F	W	—	3 ⌋ area	4 weeks
9	42	F	W	—	Ridge 5 ⌋ area	——
10	72	F	W	Yes	Ridge 5 ⌋ area	——
11	6	F	W	No	Between 1 ⌋ 1	Apparently since time of loss of A ⌋ A
12	36	F	W	—	Buccal aspect of ⌋ 6 7	3 years
13	50	F	W	Yes	Lower Bicuspid region	——
14	11	M	W	—	Buccal to ⌋ 4 5 6	1 year
15	44	M	W	—	Buccal to 7 ⌋	——
16	29	F	W	No?	Labial to 4 3 ⌋	8-10 weeks

Case #5. A fifteen year old white girl presented with a mass of one month's duration, labial to the lower right lateral incisor. This mass had increased rapidly in size until the lower right lateral incisor was almost completely covered from the labial aspect. The mass was described as "pedunculated, firmly adherent to the periosteum, with no bone between the tooth and the tumor itself." Roentgenographic examination showed no involvement of the underlying bone.

Case #6. This patient was a male (age not given) who presented with a growth labial to the socket of the upper right cuspid which had gradually increased in size since the cuspid was extracted one year previously.

Case #7. A sixteen year old white girl presented with a bluish discoloration of the gingiva in the lower incisor area which she had thought to be a bruise until about one month after she first noticed it, at which time the lower left central incisor was exfoliated. The mass was approximately 20x25 mm. in size and was firm but not hard, was very dark red with a bluish cast and the outer surface was pebbled. The mass was located entirely on the anterior mandible between the lower left lateral and right central incisor and had resulted in displacement of the teeth laterally. No X-rays were available.

Case #8. A forty-two year old white woman presented with a soft spongy mass approximately 16x6 mm. in size and irregular in shape over the crest of the upper right maxilla in the bicuspid area. She had had her teeth out for several years and has had two sets of dentures. The mass was not completely covered by bone. No X-rays were available.

Case #9. A thirty-nine year old white woman presented with a painful swelling of four weeks' duration in the upper right cuspid area. The tumor was a semi-pedunculated mass with a color varying from pink at the periphery to reddish blue in the center. The tumor measured 15 mm. in diameter, was raised 7 mm. and appeared fibrotic at the periphery. No X-rays were available.

Case #10. A seventy-two year old white woman presented with a soft elevated mass on the edentulous ridge in the region of the lower right bicuspids Roentgenographic examination revealed a crater-like bone involvement.

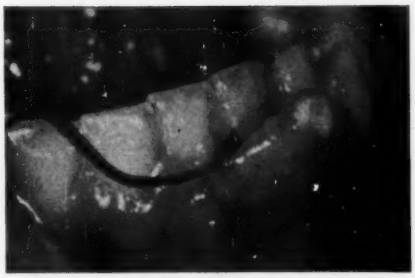

Figure 1. Typical peripheral giant cell tumor, case 1 showing pedunculation of lesion and ulceration (white area on surface). Radiographs revealed no bone changes.

Case #11. A six year old white girl presented with a mass of six to eight weeks' duration between the upper central incisors. The lesion had apparently caused separation of the incisors during their eruption as the mass had been present since soon after the loss of the upper deciduous incisors. The tumor was bluish red in color, elevated, and was situated labial, lingual and between the upper permanent central incisors. The mass blanched under pressure. The lesion recurred two weeks after initial excision, was re-excised, and to date has shown no visible evidence of recurrence. Roentgenographic examination showed no involvement of the underlying bone.

Case #12. A fifty year old white woman presented with a red, swollen mass approximately 5 mm. in diameter on the edentulous ridge in the area of the lower left first bicuspid. The patient gave a history of having had a tooth removed from this area approximately one year previously. Roentgenographic examination revealed a crater-like involvement of the underlying bone.

Case #13. A thirty-six year old white woman presented with a lesion on the buccal aspect of the upper left first and second molars. No other information is available.

Case #14. An eleven year old white boy presented with a growth on the buccal of the alveolar process extending from the upper left first bicuspid to the upper left second molar. The mass was a bony hard tumor and somewhat pedunculated. The tissue surrounding the bony part was quite friable.

Case #15. A forty-four year old white male presented with a lesion of the gingiva around the lower right second molar. The mass was 6 mm. in diameter and was similar to a fibroma in appearance. The tumor was thought to be caused by an ill-fitting partial denture.

Case #16. A twenty-nine year old white female was referred to the dental clinic because of a lesion on the labial of the alveolar ridge in the upper right cuspid and bicuspid area. The patient had been edentulous in the maxillary arch for approximately 10 years. She stated that the lesion had begun 8 to 10 weeks previously and for the last 7 days had been unable to wear her denture. The lesion was approximately 10 mm. in diameter and appeared hemorrhagic with a granular surface.

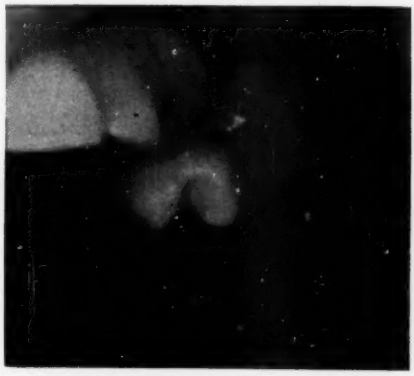

Figure 3. Large peripheral giant cell tumor in bicuspid and molar region, case 14. No radiographic evidence of bone involvement was noted.

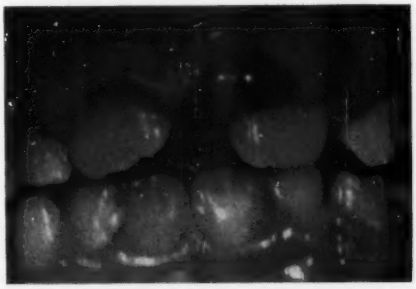

Figure 2. Peripheral giant cell tumor, case 11, in a young child. This lesion recurred following conservative surgery although no bone involvement was noted on the radiograph.

ANALYSIS OF CASE REPORTS

Of the sixteen cases available for study 50 per cent were in the maxilla and 50 per cent in the mandible. This follows the same pattern as in other reports. Thirteen of the lesions were anterior to the molar region, two in the molar region, and one

Figure 4A. Peripheral giant cell tumor in an edentulous patient, case 10.

Figure 4B. Radiographic evidence of bone involvement in this same case is noted. The "cuffing" at the periphery of the lesion is typical.

involved both the bicuspid and the molar region. It is conceivable that this latter lesion had its origin in the bicuspid region and then extended posteriorly to the molar region. This pattern of distribution supports the theory of Geschickter and Copeland[6] that the origin of the tumor is associated with the resorptive processes involved in loss of the deciduous teeth. Bernier and Cahn[8] state that, "Although the periodontal membrane ceases to exist per se after extraction of the tooth, cells with the potential for osteogenesis and cementogenesis remain." This might explain the occurrence of lesions in edentulous areas of older persons which comprised 43.8 per cent of the sixteen cases. The theory advanced by Stones[5] that the tumor might possibly arise from the undifferentiated mesenchymal cells of the periodontal membrane, seems to fall in the category of explanations for lesions that occur in the molar region and in edentulous areas of older persons. As to age distribution, 47 per cent of the lesions fell in the age group from 6 to 20, 47 per cent from 21 to 50, and 7 per cent over 50 years of age, again supporting Geschickter and Copeland's[6] theory. Although a follow-up history was not available on all patients, only one case in the present series recurred.

There were two cases in which there was a definite history of severe trauma. This is in support of the suggestion of Bernier and

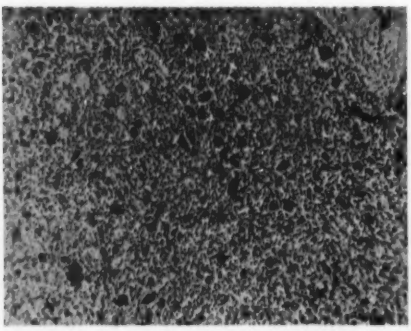

Figure 5A. A low power photomicrograph of a peripheral giant cell tumor exhibits the typical collection of large numbers of multinucleated giant cells in a highly cellular, vascular stroma.

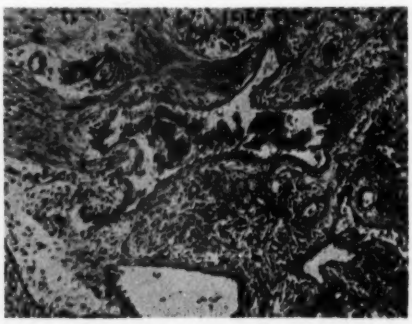

Figure 5B. A high power photomicrograph illustrates the common finding of multinucleated giant cells within vascular channels, supporting the theory of their origin from endothelial cells.

Cahn[8] that trauma is a factor to be considered as important in etiology. If this is true, then the term "peripheral giant cell reparative granuloma" is a good one, since there would definitely be some reparative reaction following an injury of this or any other type.

Most of these lesions exhibit approximately the same histologic features and are characterized by a mass of connective tissue covered by stratified squamous epithelium. Often there is an interruption of the surface epithelium with replacement by a thick exudate composed chiefly of fibrin, polymorphonuclear leukocytes and necrotic debris indicating surface ulceration which may or may not be detected clinically. The connective tissue underlying the epithelium is composed basically of interlacing collagenous fibers interspersed by varying numbers of ovoid cells resembling fibroblasts and multinucleated giant cells of the osteoclast type which may contain only a few or many nuclei. The number of giant cells varies in different lesions, in many cases a large number, while in others only a few are to be found. Varying amounts of hemosiderin pigment are also usually present, much of it within macrophages. Generally the picture is one of high vascularity which explains the dark red color of the lesion as it is observed clinically. Giant cells are often found to be numerous about the vascular channels and in some cases are found within the vascular channels which may be the basis of some earlier investigators thinking the lesion to be malignant. Also this is likely the basis for some writers believing that the giant cells originate from the endothelium of the blood vessels. Some sections show areas of hemorrhage which may have resulted from trauma to the lesion by biting into it with the opposing teeth or by traumatizing it.

Some writers classify the lesion into early, mature, and retrogressive tumors using as a basis the fact that histologically, some lesions show many capillaries and an abundance of connective tissue cells with few giant cells indicating a young growth; other sections may show numerous giant cells associated with the capillaries and collagenous fibers indicating a mature growth and the retrogressive lesion may show few giant cells but an abundance of collagenous fibers, residues of hemorrhage and hemosiderin pigment.

Whether or not this type classification is of value is difficult to determine but if the lesion is in some way related to trauma and is a reparative reaction, then a histological classification such as this may be of value in tracing the progress of such a lesion.

SUMMARY

Sixteen cases of peripheral giant cell tumor are reported. Nearly half of these lesions appeared between the ages of six and twenty, while thirteen of the sixteen cases occurred anterior to the molar region with no apparent predilection for maxilla or mandible.

The term "peripheral giant cell reparative granuloma" appears to be a valid one for use in describing this particular lesion since it undoubtedly represents an attempt at repair following some type of tissue injury.

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Root Anomalies

by DUNCAN BLADES, D.D.S., *Houston, Texas*

IF any dentist begins treatment of a patient with periodontal disease without full mouth radiographs, the case described here should help convince him of the essentiality of utilizing a complete set of good radiographs before making a treatment plan:

"Mrs. J. E. H., aged 24, was seen on March 28, 1953 suffering with a severe periodontal condition. Examination revealed poor oral hygiene associated with Class II malocclusion. The appearance of the gingiva was magenta, blunted papillary contours, swollen and irregular marginal contours, glossy texture, and of turgid consistency (see Fig. one). The gingival crevices averaged 3 to 4 mm in depth, and there was a purulent exudate. Radiographs revealed partial or complete absence of many tooth roots (see Fig. two). The patient reported orthodontic treatment was instituted at age 13, but the patient removed the appliances at age 16."

Obviously, any treatment plan for such a case as this must consider the limited area of attachment of the periodontal membrane on the root surface.

Figure 1.

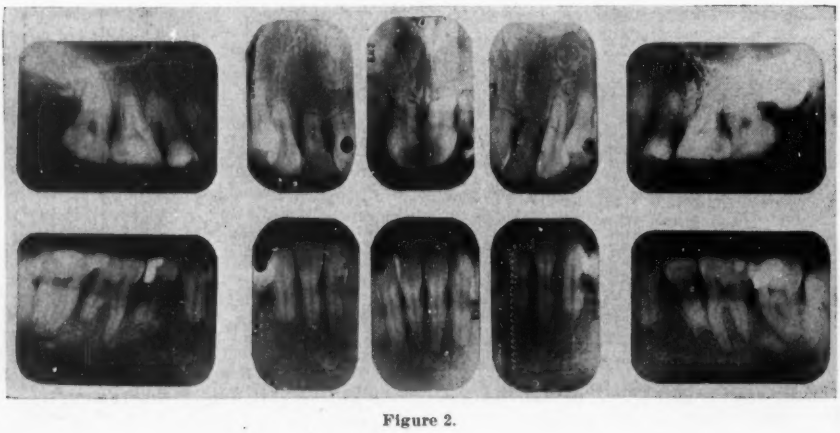

Figure 2.

DYNAMIC EQUILIBRIA OF ORAL TISSUES

III. Effects of Acute Starvation on Cytochrome Oxidase and Succinoxidase Activity of Oral Tissues

by PHILIP PERSON, D.D.S, PH.D., AND GEORGE W. BURNETT, D.D.S, PH.D.,
Washington, D. C.

IN previous studies we have examined the oxidative metabolism of rat tongue, and submaxillary and parotid gland homogenates in terms of cytochrome oxidase and succinoxidase activity and compared them with the activity of such enzymes in similar homogenates of heart, liver, and kidney.[1] It was found that the aerobic metabolism of the oral tissue homogenates was comparable to that of the homogenates of other organs whose aerobic metabolism is quite active. In extending the investigation of the metabolism of oral tissues, we wished to ascertain whether or not they participate in the dynamic metabolic equilibria which maintain the integrity of the organism. A method of ascertaining such participation would be to compare the cytochrome oxidase and succinoxidase activity of control tissues with similar tissues from animals subjected to such a severe metabolic stress as is found in acute starvation. This report is concerned, therefore, with the effect of acute starvation upon the aerobic metabolism of several oral and other body tissues of adult white rats.

METHODS

For this study 49 adult, male, Sprague-Dawley rats were maintained on a diet of Purina laboratory chow pellets and water, ad-libitum, for 24 days prior to the initiation of the experiment. Purina chow pellets were used as food instead of a purified-type ration because it has been our experience in this laboratory that rats grow better on the former diet. On the 25th day, the animals were divided into two groups of 24 control, and 25 experimental rats, respectively. Thenceforth, experimental rats received only water, ad-libitum, while control rats continued to receive the chow diet. The animals were sacrificed by decapitation at 1, 2, or 3 day intervals during the course of the experiment, which lasted 16 days. The tongue, submaxillary and parotid glands, liver, heart and kidney were removed immediately after sacrificing the animal, and cytochrome oxidase and succinoxidase activity was determined by the methods described in a previous paper.[1] Oxygen utilization, indicative of enzyme activity, is reported per milligram of nitrogen per hour.

RESULTS

Twelve of the starved animals died during the course of the experiment; 13 were sacrificed for study at appropriate intervals. There were no deaths in the control animals during the 16 days of the experiment. Starved rats varied considerably in response to deprivation of food. Some animals became moribund and lethargic after 5 or 6 days; others, although severely emaciated following a longer period of starvation, moved about nimbly and appeared as alert as the control animals. There was, however, no correlation between the appearance and behavior of an animal, and enzymatic activity of its tissues.

The average maximal change in cytochrome oxidase and succinoxidase activity of the tissues studied, as evidenced by oxygen consumption, is shown in Table I.

Third of a series of articles from the Department of Dental Research, Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington, D. C.

The authors wish to acknowledge the able technical assistance of Cpl. Joseph Nardone and Pfc. Henry Szcepanik.

TABLE I

Effects of Starvation on Enzyme Activities of Tissue Homogenates, Expressed as a Percentage of the Enzyme Activity of Normal Tissues.

Tissue	*Enzyme System*	
	Cytochrome Oxidase	*Succinoxidase*
Tongue	−53 +49	O
Parotid Gland	O	−49
Submax. Gland	O	−31
Liver	−39	O
Heart	+?	−?
Kidney	O	O

− = decrease in activity;
+ = increase in activity;
O = no significant change in activity;
? = slight but not definite change in enzyme activity.

Decrease or increase in oxygen uptake was considered significant only if the change was at least three times greater or lesser (i.e., ± 30 per cent) than the experimental error of manometric determinations, which is considered to be approximately 10 per cent. Cytochrome oxidase activity was not significantly affected in parotid and submaxillary gland, heart, and kidney homogenates; its activity was decreased in liver while its activity in tongue homogenates was decreased during the first ten days, then increased during the last six days of starvation. Succinoxidase activity of tongue, liver, heart, and kidney homogenates was not significantly affected during the 16 days of starvation. On the other hand, the succinoxidase activity of parotid and submaxillary glands was significantly decreased during this time.

The various oral and body organs responded to starvation in three principal ways. In the first type of response, there was no significant difference between the enzyme activity of the tissue homogenates of starved and control animals. Thus, neither the cytochrome oxidase activity of parotid and submaxillary glands, and kidney, nor the succinoxidase activity of tongue, liver, and kidney were affected significantly by starvation during this experiment. This type of response is illustrated in Figure 1 which shows the effect of starvation on the succinoxidase activity of tongue homogenates. With the exception of one starved animal, sacrificed on the first day, there was no significant change in succinoxidase activity of tongue from either the control or starved groups of animals during the course of the experiment. The single instance in which succinoxidase activity decreased is probably a real phenomenon, rather than an artifact, for it is an example of individual variation so constantly encountered in animals and humans. Since the factors involved in such individual variations were not determined in this instance, they cannot be discussed but attention is called to their existence.

In the second type of enzyme response to starvation, there was an initial decrease followed by an increase in activity considerably above that of similar tissues from control animals. This phenomenon is illustrated in Figure 2, in which is shown the effect of starvation on the cytochrome oxidase activity of tongue homogenates. During the first six days of the experiment, cytochrome oxidase activity of the tissues of starved animals was 53 per cent less than that of the tissues of control animals. Again, we encounter the phenomenon of individual biological variation in the tissues from the starved animal sacrificed on the

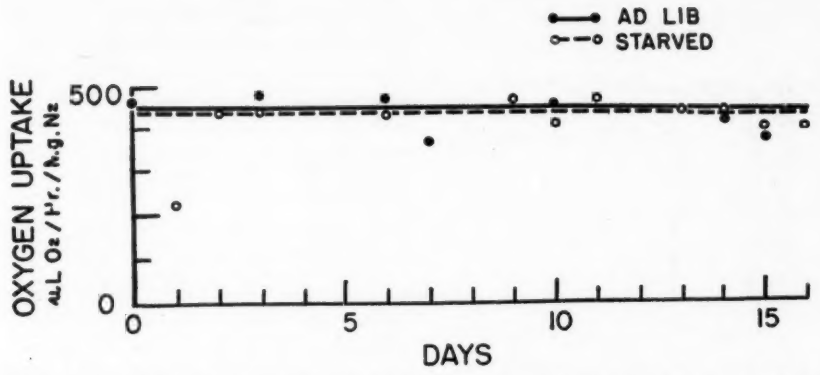

Fig. 1. A comparison of the succinoxidase activity of tongue homogenates from starved and normal animals, showing no change in enzyme activity.

tenth day. With the exception of a single variable animal, after the tenth day the cytochrome oxidase activity of the tongue tissues of the starved animals was 49 per cent greater than the activity of similar tissues from animals on the ad-libitum chow diet.

In Figure 3 is shown the third type of enzyme response to starvation. There was a steady decline in cytochrome oxidase activity as starvation progressed, resulting in 39 per cent less activity in the tissue homogenates of starved animals than in similar tissues from normal animals. The slope of the curve of enzyme activity is drawn somewhat arbitrarily for liver homogenates of starved animals sacrificed during the last

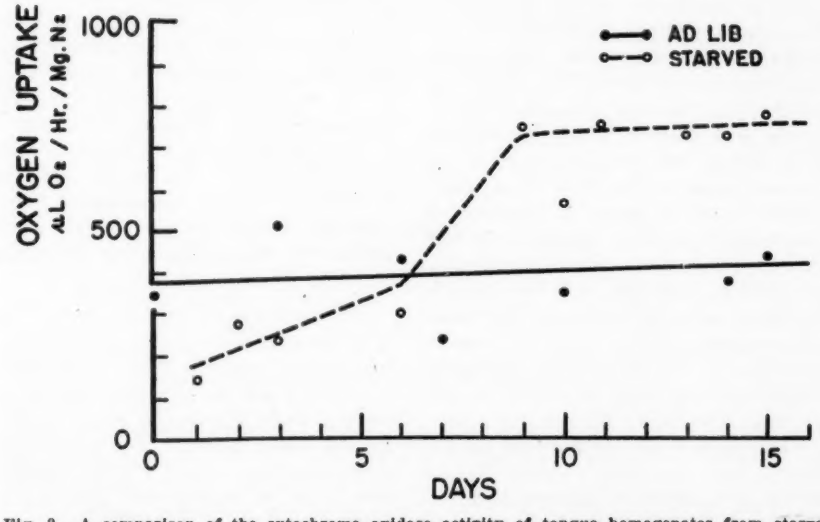

Fig. 2. A comparison of the cytochrome oxidase activity of tongue homogenates from starved and normal animals, showing an initial decrease followed by an increase in activity in the tissues from starved animals.

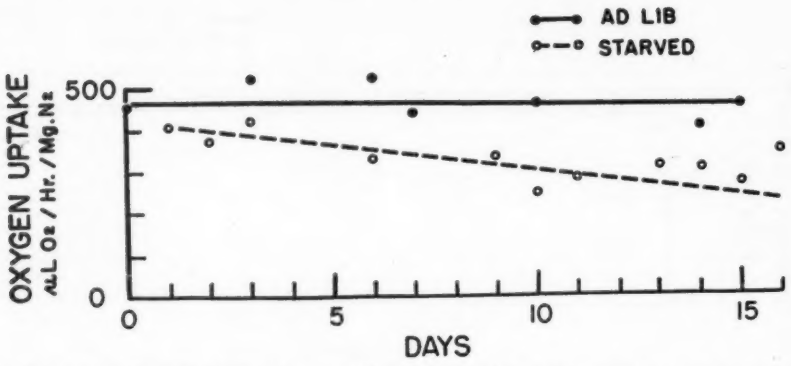

Fig. 3. A comparison of the cytochrome oxidase activity of liver homogenates from starved and normal animals, showing a gradual decrease in activity of the tissues from starved animals.

5 days of the experiment, for it is difficult to determine from the values obtained whether the decline in activity would continue or whether it was stabilized at a level below that of normal tissue homogenates. A reduction in succinoxidase activity was observed in parotid and submaxillary gland homogenates in which the decreases were 49 per cent and 31 per cent respectively.

DISCUSSION

Under conditions of this experiment, cytochrome oxidase and succinoxidase activity of tissue homogenates responded in three ways to starvation. There was either no change, a decrease followed by an increase, or a decrease in enzyme activity. It is obvious that the oral tissues of the rat participate in the shifts in dynamic equilibria which occur in response to starvation. It is very important to note that the response of a tissue to starvation may change with time, as is illustrated in Figures 2 and 3. For this reason it is necessary that the enzyme activity of tissues be determined at periodic intervals during the course of starvation, as well as at the end of such treatment. Of the six tissues studied, the enzyme activity of tongue, and parotid and submaxillary glands changed under the influence of starvation, while of the non-oral tissues, only liver was definitely affected by such treatment during the time period of this experiment. The question now arises as to whether these changes are the result of actual loss or gain of specific enzyme moieties or because of other reasons. A decrease in oxygen uptake by a tissue homogenate might be the result of a generalized loss of cell protein, or it might represent loss of specific enzyme protein, with other cell proteins remaining relatively unaffected. Similarly, an increase in oxygen consumption might represent an actual increase in amount of the enzyme, or it might indicate that enzyme protein is conserved as other cell proteins are lost during starvation. The question is further complicated by the possibility that changes in amount of regulators (i.e., inhibitors or activators) of the enzymes cause the decrease or increase in activity. The specific effect of starvation on cytochrome oxidase and succinoxidase activity in terms of the above possibilities, will be determined by further experimentation.

The thesis was developed previously that oral tissues are linked, by means of enzymatic processes, to the dynamic metabolic equilibria which maintain the integrity of the organism.[2] It was also suggested that specific intracellular enzyme proteins, which play key roles in cellular economy may be lost with cytoplasmic protein during disease or starvation. An implication, now restated, is that many biochemical

and morphological lesions are the direct sequelae of changes in enzymatic equilibria caused by the loss or changes in activity of specific enzyme proteins. These views and concepts are not original with us, since their validity in other tissues has been demonstrated by the classic studies of R. A. Peters and co-workers of the biochemical lesion of thiamin deficiency[3] and the more recent work of G. Cori on the enzymatic lesion of liver glycogen disease.[4]

In previous studies we have demonstrated considerable aerobic metabolic activity in such oral tissues as rat tongue, and submaxillary and parotid glands. The variables involved in the techniques of measuring enzyme activity were thoroughly analyzed so that reproducible and near optimal methods might be used in subsequent experimentation. In the present study, these methods have been employed to demonstrate that oral tissues do participate in the metabolic changes accompanying starvation, through important enzymatic mechanisms.

SUMMARY

1. The effect of starvation upon cytochrome oxidase and succinoxidase activity of homogenates prepared from rat tongue, submaxillary and parotid glands, liver, heart, and kidney, has been studied by manometric methods.

2. Based upon oxygen uptake per hour per milligram of tissue nitrogen, there was no detectable change in parotid and submaxillary gland, heart, and kidney cytochrome oxidase activity and no detectable change in tongue, liver, heart, and kidney succinoxidase activity.

3. There was a decrease in activity of liver cytochrome oxidase, and parotid and submaxillary gland succinoxidase.

4. Tongue cytochrome oxidase first decreased and then increased in activity.

5. It was concluded that the findings indicate that oral tissues participate in the dynamic equilibria which maintain the organism.

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ROWE SMITH MEMORIAL FOUNDATION

The Rowe Smith Memorial Foundation announces its second annual seminar, to be held December 4, 5, 6 and 7, 1955 in Karnack, Texas. The subject will be The Physiology and Pathology of Bone as Applied to Dental Practice.

For details write Rowe Smith Memorial Foundation, 501 State National Bank Building, Texarkana, U. S. A.

Editorial • • • •

SHOULD ACADEMY MEMBERSHIP REGULATIONS BE CHANGED?

As has been true in many other organizations, when the American Academy of Periodontology was young, leaders were active in searching out dentists for membership, and the only qualification for membership was that the individual be an ethical dentist with the sincere desire to learn more about periodontology. Eventually, the Academy reached such a size that it became necessary to consider methods of limiting membership to prevent the organization from growing until it became so large that many of the virtues of the Academy as it has existed in the past, would be lost. The philosophy then began to be expressed that membership in the American Academy of Periodontology should be limited to those who were especially qualified in the field of periodontology. This point of view has been challenged by many.

Recently the Journal published a statement from the Membership Committee outlining regulations regarding new members as set up by the Membership Committee.[1] Actually, these regulations go beyond those given in the Constitution and Bylaws, in some respects. However, the Membership Committee is given the power "to conduct a program, subject to the direction of the Executive Council, for the maintenance of membership . . . . . ."[2]

After the statement was published, several members of the Academy challenged the plan of controlling membership and asked to have space in the Journal to defend the point of view that membership should not be too limited. It is tempting to the Editor to publish these comments, but it is true that they would consume much space and probably little good would result. It is the request of the Editor, therefore, that all individuals who may be unhappy with the present membership regulations as set up by the Membership Committee, write the Editor or some member of the Council, with the request that this matter be thoroughly reviewed at the next meeting of the Executive Council. If a majority of the membership is unhappy with the regulations of the Membership Committee, the Executive Council should take steps to change them.

[1]Journ. of Periodont., 26:41 (January) 1955.

[2]Bylaws of American Academy of Periodontology. Sec. 14, paragraph b part 2, page 17.

The Distribution of Mitoses in the Epithelium of the Interdental Papillae of the Rat Molar*

by CHRISTINE M. HIRT, ST. HARTL AND HANS R. MÜHLEMANN, *Zurich, Switzerland*

THE structural changes occurring in the intermolar rat papilla subsequent to the begin of tooth eruption until the third year have been described in detail by Skillen.[1] The sequence of these changes were:

1. Proliferation of the oral epithelium on the connective tissue side of the epithelial attachment (diagram I).

2. Atrophy of the epithelial attachment in the process of which it slowly assumes the appearance of a keratinized epithelium.

3. Keratinization of the surface of the proliferated oral epithelium.

4. Separation of the mouth epithelium from the atrophied epithelial attachment.

5. Downgrowth of the epithelial attachment of the cementum.

In a careful interpretation of the results, Skillen does not decide whether the epithelial attachment (enamel epithelium) having fulfilled its function, degenerates first, this making it necessary for the mouth epithelium to cover over a potentially open surface,[2] or if the proliferating oral epithelium by some unknown mechanism is including degeneration and atrophy of the tissue.

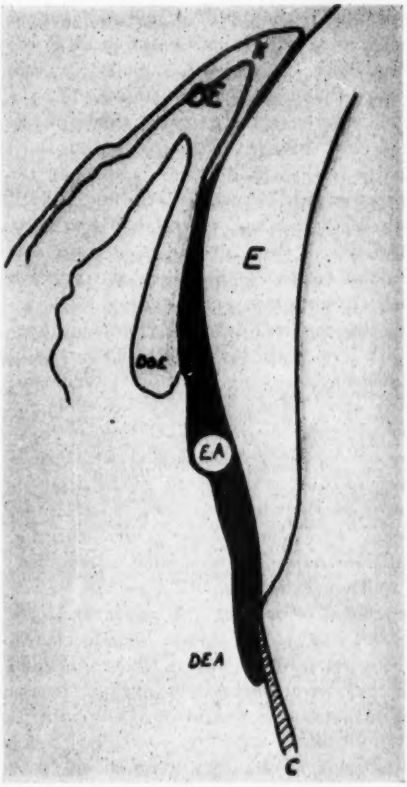

Diagram I

OE—Oral Epithelium. K—Keratinized Layer of OE. DOE—Downward Growing OE. EA—Epithelial Attachment. DEA—Downward Growing EA. C—Cementum. E—Enamel.

Since both proliferation and degeneration may be evaluated by the mitotic activity of the epithelial cells, a study of the distribution of mitoses in the oral epithelium (OE) and the epithelial attachment (EA) seemed to be of some interest. It is assumed (1) that the presence of abundant mitotic figures within a tissue may be interpreted as the equivalent of proliferation, (2) that the presence of abundant mitotic figures within a tissue may not be interpreted as tissue degeneration or atrophy.

The pattern of mitoses distribution could give some additional information about the dynamic relationship between the oral epithelium and the epithelial attachment.

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The authors wish to express their acknowledgement to Dr. C. M. E. Eugster, D.D.S., L.D.S., R.C.S. for assistance in translation.

MATERIALS AND METHODS

The materials and histologic method for this study were the same as those used in a report about the mitotic activity in the periodontal tissues of the rat molar.[2] Epithelial mitoses were located and counted in sections of 60 intermolar papillae belonging to 30 male rats, 5 months of age. Since the morphologic outline of the papilla between the first and second molar and that between the second and third molar is slightly different, and since the direction and depth of the mesiodistal histologic section may also somewhat modify the shape of the papillae, it was decided to divide the epithelial structures into characteristic regions (diagram II): tip of the papillae, downward growing oral epithelium, epithelial attachment and downward growing epithelial attachment. The *oral epithelium* (OE) was differentiated from the *epithelial attachment* (EA) by its slightly deeper staining with hematoxylin-eosin, and by the closer arrangement of the squamous cells. Furthermore there was generally more intraepithelial inflammatory cell infiltration in the EA. Papillae in which the OE could not be distinguished from the EA were discarded. It was also noted if mitoses were located in the basal layer or within the epithelium, if they were immediately adjacent to the enamel or to the cementum.

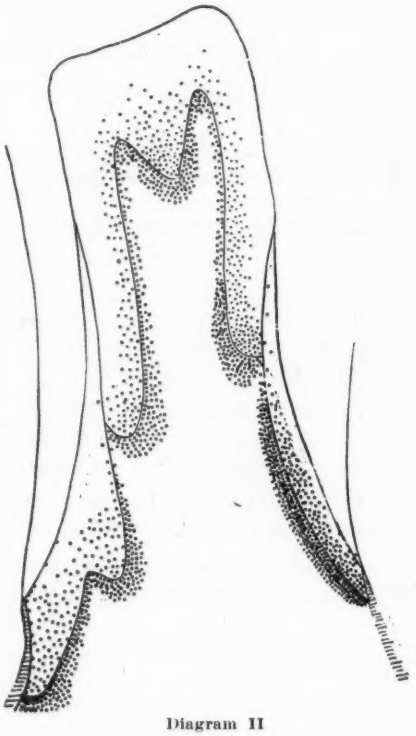

Diagram II

Composite papilla composed of 60 intermolar papillae. Distribution of 1601 mitoses. Mitoses of the basal layer are represented in the subepithelial connective tissue.

RESULTS

The distribution of 1,601 mitoses found is represented in diagram II, where the characteristic regions of the papillary epithelia are assembled to a composite papilla. Mitotic figures were found all over the papillary epithelium in the basal and prickle cell layers. They are most abundant in the downward growing OE but also within the downward growing and not downward growing apical regions of the EA. In the distal EA where downgrowth was occurring more often than on the mesial, mitotic figures were also located immediately adjacent to the root cementum. In the EA eight dividing cells with their long axes parallel to the enamel surface could be detected in close vicinity to the enamel-matrix, but this only in the cervical region of the enamel.

There were considerably less mitoses in the zone where the downward proliferating OE joins with the EA. Cell division in the EA increased steadily in frequency from the occlusal to the apical. In the EA there were only a few mitoses in the neighborhood of the downward proliferating OE. No mitoses were found in the most superficial layers of the latter structure.

DISCUSSION AND CONCLUSIONS

If abundance of cell division is a means to distinguish between proliferation or

atrophy of epithelial structures, then epithelial atrophy is only likely to occur at the junction between the downward proliferating oral epithelium and epithelial attachment. There is no indication that those regions of the EA being some distance from the OE are areas of degenerating or atrophying epithelium. Our findings demonstrate that there is an atrophic or degenerating zone at the level of the junction between OE and EA confirming the contention of Becks[3,4] that the OE by downward proliferation at the connective tissue side of the EA is successively replacing the degenerating EA.

Proliferation of the *enamel epithelium "prickle cells"* prior to tooth eruption has been described on human autopsy[5] and experimental Rhesus monkey material.[6] Skillen[1] and Belting[7] reported extensively about the downward growth of the epithelium attachment in the rat molar. Proliferation was interpreted to occur in these reports by the observation of encroaching penetration of enamel epithelial prickle cells between ameloblasts,[4] or by the description of topographic changes of the epithelial structures when evaluated in longitudinal studies.[1,5] The observation of the mitotic activity* and the distribution of mitoses gives a clue for the detection of dynamic processes of growth and physiologic repair in areas which with conventional histologic methods are more uncertain to be evaluated in respect to their "vitality." Thus it was rather astonishing to observe pronounced mitotic activity within the EA adjacent to the cervical region of the enamel. However no conclusions can be drawn as to whether these processes of cell division are the expression of a metaplastic transformation of the attached reduced enamel epithelium into a stratified oral epithelium.[9-11] Another finding of interest was the distribution of mitoses in the downward growing epithelial attachment. Mitoses regularly were found in the basal layer, within the epithelium, and also in close relationship to the cementum. The latter finding points to the mechanism of the mode of attachment of the downward proliferating epithelium to the cementum. Whatever the biophysical character of the adhesion to the cementum may be, it is reasonable to assume that living and proliferating cells are involved during the formation of the epitheliocemental attachment. Orban,[12] and more specifically Baume,[6,13] have shown the role of tonofibrils in the attachment and cohesion of neighboring biostructures. Tonofibrils are fibrous proteins chemically different from keratinized fibers[14] and are only formed in the cytoplasm of living epithelial cells. The fact that cell division was taking place not only in the basal layer,[12] but also in direct contact with the cementum, makes it probable that in the first phase at least, the attachment of the epithelium to the cementum can only be realized by non-keratinized elements.

The presence of mitoses in the immediate neighborhood of the cementum was in contrast to the very few dividing cells found in the epithelium directly facing the enamel. This observation—made on rat material—may be a contribution in the discussion of the much argued character of the human "epithelial cuff."[15]

SUMMARY

The topographic distribution of 1,601 mitoses found in 60 intermolar papillae of 30 male rats, 5 months of age, was described. Mitotic figures were found in the oral epithelium as well as in the epithelial attachment, but only exceptionally in the areas where these two epithelial structures join. In the downward proliferating epithelial attachment cell division was observed in close contact with the cementum surface. However, in the epithelial attachment, mitoses were practically never present in the most superficial layers adjacent to the enamel surface.

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*A report about the quantitative evaluation is being prepared.[8]

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"Irvangeline"

A Symphony in Bone*

This is the jargon primeval
The murmuring polysyllabics
Eponyms German and English
Far worse than the poets iambics.

Osteoclasts christened by Kolliker
Lurking in Howship's lacunae
Active or passive in function!
Argued from then until doomsday.

Havers and systems Haversion
Woven in intricate patterns
Viewed with crossed Nicols, inversion,
Shunned by scholastic slatterns.

Pierced by Volkmann's canals
Joining the systems Haversion
Studied with zealous endeavor—
Remembered with utter aversion.

These the trabecular patterns
Functional stresses reflecting
Obeying the laws of Wolff
Object of student neglecting.

Periosteum and fibers of Sharpey
Endosteum and marrow poietic
Mineralized matrix immortal!
Oh, but I'd rather forget it.

And remember the Berkshire mountains
The silence of lake and of soil
Untroubled by factors and functions
Expounded by Glickman and Boyle.

*Written with apologies to H. W. Longfellow, by Dr. Paul A. Boyle, for the Berkshire Conference. Director, Dr. Irving Glickman.

The Relationship of Blood Catalase Activity and Periodontal Disease*

by HAROLD R. ENGLANDER;[1] IRVING L. SHKLAIR;[2] R. QUENTIN BLACKWELL,[3] AND LEONARD S. FOSDICK[4]

IN 1946 provocative observations were made in Japan on several cases of progressive oral gangrene in children. The reports of these cases[1,2] prompted the initiation of this study.

In 1946, Shigeo Takahara, an otolaryngologist practicing in Japan, treated an 11 year old girl who had recurrent gingival ulcers and a 3 year history of tooth exfoliation. The most conspicuous oral symptom was a peculiar progressive oral gangrene starting in the maxillary gingivae and extending to the antrum and nasal cavity. Severe alveolar bone loss was evident with concomitant mobility of remaining teeth. The patient was treated by broad excision of the affected parts and during the course of operation, the debrided areas were lavaged with hydrogen peroxide. The wound surface immediately exhibited a brownish-black discoloration instead of the usual vigorous foaming action. Subsequently, it became apparent that the absence of catalase in the girl's blood was directly responsible for this phenomenon. In normal blood the presence of catalase causes the decomposition of hydrogen peroxide, $2H_2O_2 \xrightarrow{\text{Catalase}} 2H_2O + O_2$. In this acatalasemic girl, lack of catalase prevented the decomposition of hydrogen peroxide with the resultant oxidation of the blood to the brownish-black methemoglobin.

Three other children (2 girls and 1 boy) in this family of six siblings exhibited acatalasemia. All 3 exhibited this progressive oral gangrene with loosening and exfoliation of teeth at some time during their childhood. After complete removal of teeth, further symptoms of gangrene did not occur. Two more Japanese families were found in which children exhibited acatalasemia. The "only child" in one family exhibited severe symptoms at 13 years of age with ulceration of tonsillar crypts and ulceration of the tongue from direct extension of a gingival lesion. Four siblings in the other family of 10 children manifested acatalasemia. Two of these 4 had slight gingival symptoms during childhood.

Since 1946, seven more corroborative cases of acatalasemia have been reported in Japan making a total of 16 cases in all. Fourteen of 16 (88%) showed this peculiar progressive type of noma at some time during childhood. Serious systemic symptoms were checked by adequate treatment in all cases.

Takahara et al[3] determined the Quotient of catalase of nasal and oral tissues and blood. The Quotient of catalase of the oral and nasal tissues in the acatalasemia cases was always zero. When the ratio of the blood Quotient of catalase of the cases was compared to the normals, it ranged from 1/500 to 1/1000. No noticeable evidence of change in the catalase levels of the blood in the acatalasemia cases occurred over a period of 5 years.

None of the cases reported by Takahara gave histories of receiving atomic bomb or x-ray radiation and many cases came from widely separated prefectures in Japan. It was noted that consanguineous

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The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Department of the Navy, or the Naval Service at large.

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[2]Bacteriologist, Dental Research Facility.

[3]Assistant Professor of Chemistry, Northwestern University.

[4]Professor of Chemistry, Northwestern University.

marriages occurred in the parents of the original 9 cases and suggested that this condition may be inherited as a Mendelian recessive characteristic.

A possible explanation for the necrosis was given by the otolaryngologist. He claimed that the hemolytic streptococcus and pneumococcus isolated produced hydrogen peroxide. As these organisms proliferated in tissues traumatized by mastication, hydrogen peroxide was generated which oxidized the blood to methemoglobin thus depriving the injured tissue of available oxygen. This was born out microscopically by the lack of inflammation in the necrotic tissue examined.

On the basis of the severe alveolar bone loss associated with this condition, it was decided to initiate a study on the relationship between catalase activity in the blood and periodontal disease.

PROCEDURE

The rapid and accurate method of Landahl[4] for the catalase determinations was used. The method is based upon the fact that heat is evolved when H_2O_2 is decomposed to water and oxygen. The rate of heat evolution and subsequent temperature rise in a tube containing a solution of H_2O_2 and catalase is proportional to the amount of catalase present. By measuring the amount of temperature rise under controlled conditions, one can estimate, very simply, the amount of catalase present.

One hundred and ninety-eight normal individuals and 59 cases of periodontal disease were studied. Subjects were selected from three sources: Navy personnel at the Great Lakes Naval Training Center, dental students and cases of extreme periodontal disease at Northwestern University Dental School Clinic. All patients were examined clinically and roentgenographically for periodontal disease. The individuals with periodontal disease manifested deep pocket formation, severe alveolar bone loss and extreme tooth mobility. The average age of the 198 normal individuals was 24 with a range of 18 to 46 years. Twenty-two per cent were female. The average age of the 59 periodontal cases was 40 and ranged from 20 to 60 years of age and forty-four per cent were female.

Duplicate determinations were made on 0.02 ml of blood drawn from each individual and the average of the two determinations used for each subject. Each sample of blood was added to a plastic tube containing 4.50 ml of pH 7.0 phosphate buffer solution and 1 drop of octyl alcohol. The temperature of each tube was brought to 25° C ± 0.5° C, the exact temperature recorded, and 0.5 ml of 30% hydrogen peroxide at 25° C was added. The temperature rise was measured by the use of a maximum temperature recording thermometer with a scale range of 25 C° — 40° C calibrated in 0.1° C intervals.

RESULTS

The results of this study are shown in Table I. The average temperature rise of 198 normal individuals and 59 persons with severe periodontal disease is 10.7° C and 10.4° C respectively with a standard

TABLE I

Mean temperature rise (°C) in blood of 198 individuals with healthy periodontium and 59 with severe alveolar bone loss

	Normal Individuals	*Periodontal Cases*
No.	198	59
Av. Temp. Rise	10.7	10.4
σ	1.8	1.9
Range	6.7—15	5.7—14

$$\frac{\text{Deviation}}{\sigma} = \frac{(\bar{x}_n - \bar{x}_p) - 0}{(\sigma_{\bar{x}_p} - \bar{x}_p)} = 1.1$$

(Not statistically significant. The probability of equalling or exceeding 1.1 standard deviations away from zero is 27 per cent or less.)

deviation of 1.8° C and 1.9° C. The normals ranged from 6.7° to 15.0° C and the periodontal cases from 5.7° C to 14° C. The distribution for both samples was well balanced. The difference of 0.3° C between the means of normals and periodontal cases was tested statistically and was not found significant.

CONCLUSIONS

There does not appear to be any difference in the blood catalase activity of individuals with normal periodontal structures and those with severe periodontal disease. A normal range of blood catalase activity has been established by this method and ranges from a 10° C to 11° C rise in temperature and corresponds favorably with the results of Landahl. The close reproducibility of results on duplicate samples from each individual supported the method used for catalase determinations.

SUMMARY

1. The rationale behind this study based on acatalasemia in Japan was described.

2. To date, 16 cases of "acatalasemia" have been reported by other investigators in Japan. Fourteen of sixteen (88%) showed a peculiar and characteristic progressive oral gangrene during childhood. No cases have been reported in the U.S.A.

3. There was no difference in the blood catalase activity of 198 normal and 59 individuals exhibiting severe periodontal disease.

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AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-ANNUAL MEETING

The American Academy of Dental Medicine will hold its Tenth Mid-Annual Meeting and Luncheon at the Hotel New Yorker, New York City, on Sunday, December 4, 1955, 12:30 P.M. The program includes a symposium on THE ORAL SOFT TISSUE IN HEALTH AND DISEASE. Participating will be Dr. Edward V. Zegarelli of New York City, Dr. Hamilton B. G. Robinson of Columbus, Ohio, and Dr. Lester W. Burket of Philadelphia, Pa.

All members and interested dentists and physicians are cordially invited to attend. For reservations and programs, address the National Secretary, Dr. George J. Witkin, 45 South Broadway, Yonkers 2, N. Y., or the Chairman, Dr. Alonzo N. De Vanna, 3063 Buhre Avenue, New York 61, N. Y.

Announcements • • •

TENTATIVE PROGRAM OF THE 1955 MEETING OF THE AMERICAN ACADEMY OF PERIODONTOLOGY

Sheraton-Palace Hotel, San Francisco, California — October 13, 14, 15, 1955

THURSDAY

10:30-11:00 Presidential Address
Harold G. Ray, D.D.S., San Francisco

11:00-11:45 (Title still open)
Willard C. Fleming, D.D.S., San Francisco

12:00- 2:00 Lunch

2:00- 2:50 Integrating Various Fields of Dentistry with Periodontics—A Symposium
Operative Dentistry: Gerald D. Stibbs, D.D.S., Seattle, Wash.
Orthodontics: Ernest Johnson, D.D.S., San Francisco
Crown and Bridge Prosthesis: Allison James, D.D.S., Beverly Hills, Calif.
Denture Prosthesis: George Hughes, D.D.S., San Francisco

2:50- 3:15 General Discussion
Moderator—B. O. A. Thomas, D.D.S., Seattle, Wash.

3:20- 5:00 Treatment Planning Clinic
Moderator—Harold Ray, D.D.S., San Francisco
Presentation of practical case problems by the above panel plus—
Radiography: Theodore Grant, D.D.S., San Francisco
Periodontics: Harold Ray, D.D.S., San Francisco

FRIDAY

9:00-10:15 Evaluation of Research Findings Relative to Clinical Application—A Forum: Under direction of the Research Committee
Irving Glickman, Chairman, Boston, Mass.

10:30-11:15 The Influences of Adrenal Gland Substances (ACTH, Cortisone, etc.) on the Defense Mechanism
Edgar S. Gordon, M.D., Madison, Wis.

11:15-11:30 Discussion

11:30-12:00 Bacterial Flora of
(a) The newborn and infant child's mouth
(b) The normal adult gingival crevice
(c) The pathological periodontal pocket
Valerie Hurst, Ph.D., San Francisco

12:15- 2:00 Round table luncheon
Justin D. Towner and R. Gordon Agnew, Co-Chairmen
Speaker: Burton Meyer, Ph.D., Radiation Laboratory, Berkeley, Calif.

2:15- 3:00 Current Knowledge of Nutrition Relative to the Supporting Dental Tissue
Sam Dreizen, D.D.S., Chicago, Ill.

3:00- 3:15 Discussion

3:30- 4:00 Epithelial Attachment
Balint Orban, D.D.S., Colorado Springs, Colo.

4:00- 4:15 Discussion

4:15- 4:50 Periodontal Surgery
A. W. Ward, D.D.S., San Francisco

4:50- 5:00 Discussion

SATURDAY

9:00- 9:30 Osteoectomy
Nathan Friedman, D.D.S., Beverly Hills, Calif.

9:30-10:30 Nutrition & Metabolism in Periodontal Disease—Round table discussion
Sam Dreizen, D.D.S., Chicago, Ill.
Edgar S. Gordon, M.D., Madison, Wis.
Eugene Ziegler, D.D.S., Los Angeles, Calif.
Henry Leicester, Ph.D., San Francisco
Moderator: Maynard K. Hine, D.D.S., Indianapolis, Ind.

10:30-11:00 Clinical Procedures in Periodontal Therapy
Dickson Bell, D.D.S., San Francisco

11:00-12:00 Business Session

Saturday afternoon—Clinics program

Dr. Martin Cattoni, University of Texas: "Phase-Contrast on Vincent's Infection."

Dr. Robert L. Roseberry, San Francisco: "The Surgical Eradication of the Periodontal Pocket."

Dr. Erwin Schaffer, Camp Pendleton, California: "Indications and Clinical Procedures for Subgingival Curettage and Reattachment."

Dr. E. R. Romine, Detroit: "Operative Dentistry and Periodontia."

Dr. Arnold Ariaudo, San Diego, and Dr. Robert Reeves, University of Southern California: "Periodontal Splinting: Theory, Design and Construction."

Dr. John E. Rhoads and Dr. Herbert C. Ward, College of Physicians and Surgeons: "Bite Analysis—Its Technic and Application in Periodontics."

ANNUAL MIDWEST SEMINAR OF DENTAL MEDICINE

September 11-15, 1955

The Midwest Seminar of Dental Medicine announces its Eighth Annual Seminar, to be held September 11-15, 1955, at Maxwelton Braes, Baileys Harbor, Wisconsin. This resort is situated in picturesque Door County, and is taken over exclusively by the Seminar for that period.

Purpose: Study and Relaxation

This seminar has been organized in response to the growing interest of the dental profession in informal study and discussion of biologic problems which are basic to an intelligent understanding of the health and disease of the oral structures and their treatment.

Instruction and discussion will be confined to the forenoons and evenings. The afternoons will be free from any schedule and available for relaxation and rest or for informal discussion with the faculty, according to the individual desires of each member of the seminar.

	BIOLOGY OF CONNECTIVE TISSUE (with special emphasis on the oral structures)				PROBLEMS IN COMMUNICATION
FACULTY	Paul Weiss, Ph.D. Rockefeller Institute for Medical Research	Hubert R. Catchpole, Ph.D. University of Illinois	Milton Engel, M.S., D.D.S. University of Illinois	1:00-5:00 P. M.	Irving J. Lee, Ph.D. Northwestern University
	9:00-10:00 A. M.	10:00-11:00 A. M.	11:00-12:00 A. M.	RECREATION	5:00-6:00 P. M.
Monday Sept. 12	Basic Considerations on Tissue Development	Structure of Connective Tissue	Relation of Connective Tissue to Blood and Blood Cells		Principles of Dentist-Patient Communication
Tuesday Sept. 13	Development and Regeneration of Connective Tissue	Liability and Plasticity of Connective Tissue	Formation and Resorption of Bone		Working Techniques in Communication
Wednesday Sept. 14	Functional Adaptation in Connective Tissue	Collagen Disease	Behavior of Some Specialized Connective Tissues of Oral Cavity		The Human Dimension in Dentist-Patient Relationship
Thursday Sept. 15	Summation and Round Table Discussion				

For further information write to: Dr. Isaac Schour, Executive Director, 808 S. Wood St., Room 1161, Chicago 12, Illinois.

TUFTS UNIVERSITY
SCHOOL OF DENTAL MEDICINE

Tufts University School of Dental Medicine, Division of Graduate and Postgraduate Studies, announces the following courses in postgraduate periodontology for the 1955-56 academic year.

DPG. 601—PARTICIPATION COURSE IN PERIODONTOLOGY

Monday through Friday for One Week from

November 7 through 11, 1955

Tuition $150 — Class limited to 12

Dr. Irving Glickman & Staff

Half of the time of the course is devoted to clinical practice by the participants. The remainder of the curriculum consists of lectures and seminars devoted to the theoretical and didactic aspects of periodontology. The histopathology of periodontal disease and the "Bone Factor" concept will be presented. The clinical management of periodontal disease is analyzed in detail with special consideration given to the various aspects of diagnosis, determination of prognosis and treatment planning.

DPG. 602—PARTICIPATION COURSE IN PERIODONTOLOGY

Monday through Friday for One Week from

April 9 through 13, 1956

Tuition $150 — Class limited to 12

Dr. Irving Glickman & Staff

This course consists of the same content as DPG. 601.

DPG. 603—TEMPORARY SPLINTING AND MINOR TOOTH MOVEMENT IN THE MANAGEMENT OF PERIODONTAL PROBLEMS

January 16, 17, 18, 1956—Monday through Wednesday

Tuition $75.00

Dr. Leonard Hirshfeld of New York City

This course will consist of an analysis of the technics governing the use of temporary splinting in the treatment of periodontal disease. Diagnostic and technical aspects of minor tooth movements will be presented from the viewpoint of the periodontist.

The course is arranged so that the mornings are devoted to lecture and discussions and the afternoons to clinical demonstrations and the treatment of selected cases by the participants.

For further information and application write to: Director, Division of Graduate and Postgraduate Studies, Tufts Dental School, 136 Harrison Ave., Boston, Mass.

BETH ISRAEL HOSPITAL PERIODONTIA COURSES FALL 1955

The Dental Department of the Beth Israel Hospital in Boston announces two post-graduate courses in Periodontia for the Fall of 1955.

A one week course in Occlusal Adjustment will be given from September 28 to October 2nd. This course will include background material on the anatomy; histology and physiology of the periodontium and dental apparatus. Diagnostic methods and treatment procedures for occlusal adjustment will be thoroughly discussed and demonstrated in the clinic. Participants will be limited to twelve. The tuition fee is $200.00.

A two week seminar in Periodontia is scheduled for October 31, to November 11th. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations and clinics. The tuition fee is $350.00.

Co-Directors of the courses are Henry M. Goldman, D.M.D., and Bernard Chaikin, D.M.D.

For further information write to Coordinator of Education Programs, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Massachusetts.

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY OFFERS FELLOWSHIPS

Research and Teaching Fellowships are available at the College of Dentistry in all fields of research. Stipend in each instance is approximately $3,500. Application blanks may be obtained from the Committee on Research and Fellowships, College of Dentistry, New York University, 209 East 23 Street, New York 10, New York.

WESTERN SOCIETY OF PERIODONTOLOGY TO MEET IN MARCH 1956

The next annual meeting of the Western Society of Periodontology will be held in Las Vegas, Nevada in March, 1956. The meeting will extend from Friday evening to Sunday noon; the exact dates will be announced later. This meeting, as before, will be sponsored jointly with the California Academy of Periodontology and will combine a week-end of recreation with an excellent program. The speaker for this meeting will be Dr. Henry Goldman.

Further information may be obtained from Dr. Robert L. Reeves, President, 718 Wilshire Blvd., Santa Monica, California.